

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—20TH YEAR.

SYDNEY, SATURDAY, JULY 29, 1933.

No. 5.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES—	PAGE.
"Pneumonokoniosis", by W. A. EDWARDS, M.B., Ch.M.	131
"Renal Pain and Nephropexy With or Without Resection of the Renal Nerves", by M. GRAHAM SUTTON, F.R.C.S., F.R.A.C.S.	135
"The Treatment of Carcinoma of the Uterine Cervix", by ROBERT FOWLER, M.D., F.R.C.S.	144
REPORTS OF CASES—	
"Varicella and Herpes Zoster", by G. A. MURRAY, M.B., D.P.H.	148
REVIEWS—	
The Medico-Legal Aspect of Workers' Compensation	148
LEADING ARTICLES—	
The Practitioner and His Journals	149
CURRENT COMMENT—	
Adhesions in the Upper Part of the Abdomen	150
Protein Digestion and Achlorhydria	151
ABSTRACTS FROM CURRENT MEDICAL LITERATURE—	
Surgery	152
SPECIAL ABSTRACT—	
Dental Caries	154
BRITISH MEDICAL ASSOCIATION NEWS—	
Scientific	156
Medico-Political	160
Notice	161
PUBLIC HEALTH—	
International Vitamin Standards	161

OBITUARY—	PAGE.
Thomas Glen Oliphant	161
Wentworth Roland Cavanagh Mainwaring	162
Alfred William Hill	162
CONGRESS NOTES—	
The Australasian Medical Congress (British Medical Association)	162
CONGRESSES—	
American Congress of Physical Therapy	163
POST-GRADUATE WORK—	
Annual Refresher Course in Melbourne	163
CORRESPONDENCE—	
Hay Fever and Vaso-Motor Rhinorrhœa	163
The Closed Door	163
Intermittent Alcoholism or Dipsomania	164
Unusual Hæmoptysis: An Inquiry	164
Achlorhydric Anæmia	164
The Tonsil Problem	164
The Listerian Oration	165
An Economic Commentary and Diagnosis	165
PROCEEDINGS OF THE AUSTRALIAN MEDICAL BOARDS—	
Tasmania	165
CORRIGENDUM—	
The Treatment of Diabetes Mellitus	165
BOOKS RECEIVED	165
MEDICAL APPOINTMENTS	166
MEDICAL APPOINTMENTS VACANT, ETC.	166
MEDICAL APPOINTMENTS: IMPORTANT NOTICE	166
EDITORIAL NOTICES	166

PNEUMONOKONIOSIS.¹

By W. A. EDWARDS, M.B., Ch.M. (Sydney),
Honorary Radiologist, Sydney Hospital.

TONIGHT I wish to bring before you a short paper on the condition known as pneumonokoniosis.

In the time at my disposal it is possible only to touch on the main points of this subject, but I shall endeavour to outline briefly the main points in the causation and diagnosis of the condition and to make some reference to its medico-legal aspect as it concerns the medical practitioner.

Ætiology.

Pneumonokoniosis is a condition of fibrosis in the lungs, brought about by the inhalation of dust in various occupations. The main industries concerned

are rock-chopping, coal-mining, cement and pottery working, asbestos working and tool grinding. In some of these occupations the hazard is high, and in others quite low. In New South Wales the main industries which concern us are rock-chopping and coal-mining.

The risk of contracting this condition varies in direct proportion to the amount of free silica contained in the inhaled dust, the other substances in the dust being only of incidental importance.

If the silica content is high, the condition will develop far more rapidly than if the silica content is low. A previously damaged lung, whether the damage is due to tuberculosis or recurrent bronchitis, is more rapidly affected than is a previously healthy one.

Another factor depends on the individual in some way, for men are seen who, after fifty years in the most damaging dust, namely that from Hawkesbury sandstone, show no increase in the fibrous tissue of

¹Read at a meeting of the New South Wales Branch of the British Medical Association on May 25, 1933.

the lung. The same is true in the coal-mining industry and in various metalliferous mines. In brief, the condition may appear in eight years in some and be absent in workers of fifty years, yet they have worked under similar dusty conditions.

From my observations I would set a minimum period of exposure to dust in the case of susceptible individuals without previous lung history in the sandstone industry at from eight to ten years. Hawkesbury sandstone is the most damaging of our dusts and contains upwards of 80% of free silica.

Next to the sandstone workers come those engaged in metalliferous mining; they are working in ore carrying from 20% to 60% of free silica; these men may develop pneumonokoniosis after fifteen years of exposure.

Coal miners work in seams carrying a low percentage of silica in the coal, but in addition their work necessitates much cutting through stone and country rock. The coal itself may contain 0.5% to 2% of silica, but the stone may carry higher than 30%. The analysis of dust from our coal mines is surprising in its small percentage of free silica content; from the condition of the lungs seen in these workers one would suspect higher free silica figures. It has been suggested by several workers that "silicates", which occur in high percentages, have some untoward effect, but, at present, there is no proof of their harmful effect, and the general opinion of experts is against it. It would be as well, however, to consider the possibility of their damaging action as "not proven". In these workers the earliest cases are seen after twenty years of exposure.

In other parts of the world the condition apparently is established after shorter periods of work, but the above figures are accurate in regard to workers in this State, probably owing to better working and living conditions.

The foregoing figures should be of assistance to the medical practitioner in coming to a conclusion as to whether the patient is suffering from this condition or not. Sometimes one sees a diagnosis of pneumonokoniosis made when the patient has been exposed for only four or five years to sandstone dust, or for a few years to coal dust. These diagnoses are obviously wrong and lead to endless discontent among the workers and cost large amounts in litigation.

Pathology.

To arrive at a correct understanding of the causation of this pneumonokoniosis the various pathological processes involved must be investigated.

This study is also of interest as it has a close relationship to the invasion of the lung by the tubercle bacillus in individuals who are not exposed to the dust hazard in everyday life.

The dust is inhaled in various quantities and enters the respiratory tract. By far the greater portion is trapped in the secretions of the nasopharynx, bronchi and larger bronchioles, and is expectorated or swallowed; this dust does not enter

into the causation of pneumonokoniosis, as it never reaches the alveoli.

It is the finer particles, five to ten microns in size, which reach the pulmonary alveoli and which cause the main trouble. Dust particles of larger size are rarely found in the alveoli *post mortem*.

These finer dust particles in the alveoli produce local irritation with reactionary catarrhal changes and with proliferation of the alveolar cells which take on the rôle of phagocytes. The dust particles are ingested by these cells and what is known as the "dust cell" is formed. Many of these are eliminated from the lung along the bronchial system and expectorated, but a certain proportion enter the pulmonary lymphatic system and are drained from the lung by the lymph stream and thus eliminated, and no longer interest us.

Other "dust cells", after entering the lymph stream, become arrested in the lymphatic system and the silica contained in them goes slowly into solution and they become surrounded by fibrous tissue, consequently forming the "silicotic nodule". These nodules are similar to tubercles, but do not go on to caseation.

It is the cell containing the silica particle that seems particularly liable to stagnate in the lymph stream; similar "dust cells" containing coal particles pass freely through the lymphatics and are eliminated from the lung.

It is suggested that the silica particle destroys the phagocyte and causes it to remain in the lymph channels; it has also been suggested that an abundance of dust particles in the alveolus causes an overloading of the phagocyte, which falls down at its work and remains in the lymph channel. Other suggestions have been offered, but the foregoing certainly explain the phenomena and make many obscure points in this condition much clearer.

It can now be understood why some persons who work in sandstone develop silicosis in ten years whilst others may work a lifetime in the same industry and show no changes. The worker who is affected early in his industrial life may either be taking very large quantities of dust into his lungs, owing to poor ventilation, dry drilling *et cetera*, or, on the other hand, he may have an inferior or damaged lymphatic drainage system, such as occurs after previous lung affections. Men with pre-existing tuberculosis or bronchitis develop silicosis more rapidly than those with healthy lungs.

The Radiographic Appearances.

Now as to the radiographic appearances in pneumonokoniosis.

The main difficulty is in determining when a man has contracted this condition. The changes in the lung are very gradual, taking from eight to fifty years to develop, and are often well established before there is any hint of disability or ill health.

The fibrous tissue formation commences near the hilum, affecting the right side first and gradually extending through both lungs, leaving the apices

and costo-phrenic portions of the lungs unaffected until tuberculosis is superadded.

This early fibrosis is seen as an increase in markings of the lung reticulum, and is difficult to distinguish from the appearances of bronchitis, asthma, chronic cardiac disease, syphilis, and from the lung of the average city dweller of middle age. It is surprising to find the amount of marking in middle-aged workmen who have never worked in dusty occupations, and the radiologist should take every opportunity of studying large series of such cases. The so-called "stippling" is found in a large percentage of normal persons, especially when the exposures are short and the skiagrams are taken from a distance with the use of high milliamperages and low kilovoltages.

It is strongly urged that the radiologist should wait until the definite fibrotic nodule can be demonstrated before giving an interpretation of pneumonokoniosis.

Even when the nodules are well marked there may be practically no disability and no physical signs, so why diagnose a pneumonokoniosis from a slight increase in linear markings or a slight "stippling"? Too often the radiologist is misled in his interpretation of the skiagram because he happens to have learned that the subject is a miner. This "stippling" is sometimes seen in syphilitics, and a Wassermann test is insisted upon in these cases by many authorities.

It is a serious matter to label a man as silicotic on insufficient evidence, as thereafter he waits for the onset of the dread symptoms of "miner's phthisis", of which he has seen his comrades die.

In practice the diagnosis of pneumonokoniosis when the fibrotic nodule is formed is made quite soon enough.

Pneumonokoniosis has been divided into stages, but this has proved unsatisfactory, as it is impossible to state where one stage ends and another begins. It is better to classify the stages of the disease as early, marked, advanced, and silicosis with superadded tuberculosis.

For some time an attempt was made to introduce a "pre-silicotic" stage, but this has been abandoned, as most persons placed in this stage were found to be merely bronchitics or asthmatics and never developed a true silicosis.

The size of the silicotic nodule depends on the amount of free silica present in the inhaled dust rather than on the stage of the disease.

With high silica content (80%) the nodule is large, while with a percentage under twenty the nodules are very small.

The typical silicosis then is seen as a nodular fibrosis scattered almost symmetrically on the two sides, and is not a streaking or thickening along the line of the bronchi.

The differentiation from military tuberculosis from the skiagram alone is not possible, but the clinical history and examination are generally sufficient to clinch the diagnosis.

When areas of consolidation appear towards the apices it is a sign that a tuberculosis has been superadded, and the demonstration of upper lobe cavities is of importance.

In coal miners, especially those with experience in Great Britain, there is often seen a dense area in the upper lobe regions due to subpleural consolidation and fibrosis. This occurs in the form of a "plaque", and stereoscopic or oblique skiagrams will demonstrate its superficial situation. This is regarded by most authorities as a type of chronic tuberculous cirrhosis, due to the slowing down and localization of the tuberculous infection by the well known anti-tuberculous action of the coal dust.

Compensation.

This condition of pneumonokoniosis is of great importance to the men engaged in these various hazardous occupations, and the position is a complicated one, both from the medical and legal aspects. May I here point out that three Acts of Parliament dealing with this subject are in force in New South Wales: (i) the *Broken Hill Act*, (ii) the *Silicosis Act*, (iii) the *Workers' Compensation Act*, 1926.

The *Broken Hill Act* allows men who have worked in the Broken Hill mines to be paid compensation if suffering from pneumonokoniosis either alone or complicated by tuberculosis, and, in addition, men who are suffering from tuberculosis unaccompanied by pneumonokoniosis are also compensated.

This is a very generous act from the worker's point of view, and medically it is very easy to administer. The medical authority has only to satisfy itself that the man has typical radiographic appearances of pneumonokoniosis or is suffering from tuberculosis and he is withdrawn from the industry and put on the compensation list.

This procedure is simple, and the general practitioner is not liable to be called into court to give evidence as to whether the worker is incapacitated or as to whether the condition is one of pneumonokoniosis or tuberculosis. All men desiring to enter this industry must have a preliminary radiographic and clinical examination made in order to establish their physical fitness. The cost of compensation is borne equally by the mining companies and by the Government and amounts to a very large sum annually.

The *Silicosis Act* allows compensation to be paid to men working in Hawkesbury sandstone in the County of Cumberland only, and includes rock-choppers, stonemasons and sewer workers who are incapacitated by silicosis or by silicosis accompanied by tuberculosis. Under this Act a man is not granted compensation because he has radiographic and clinical signs of silicosis, but there must be some physical incapacity due to his lung condition.

These damaged workers are granted a certain weekly payment consistent with the amount of physical disability found at the medical examination. During these examinations it is frequently

noticed that men showing definite radiographic evidence of silicosis suffer no physical disability whatsoever, and many of them are quite indignant when told that they are suffering from any lung affection and insist on returning to their ordinary daily work.

One would think that, by allowing such a man to continue at work, he is being condemned to an early death, but in practice this is not the case, as it is found that even if the man is withdrawn from the industry, the condition, once well established, gradually progresses, even when no further silica dust is inhaled. This is accounted for by the fact that the lung already contains large quantities of silica, which continue to go into slow solution and which continue to stimulate the formation of fibrous tissue with gradual progression of the condition. This gradual progression has been abundantly demonstrated by observing the progress of many hundreds of men withdrawn from the Broken Hill mines.

It is rarely found that death takes place from a silicosis alone; the serious stage is reached when a tuberculosis is superadded. Such infection means rapid deterioration in health and early death.

Many men with silicosis go on for years with little effect on their general health; they are not breathless on exertion, have no cough and show no physical signs. Suddenly, however, the man develops definite disability, becomes breathless and exhausted on slight exertion, cough becomes marked and distressing, and there is rapid loss of weight. Such a change invariably means that a tuberculous affection has occurred.

The administration of this Act is very complicated from the medical point of view, for the medical authority, consisting of two physicians and a radiologist, must not certify a man as fit for compensation unless he is definitely incapacitated, and that incapacity must be assessed as a percentage disability.

Tuberculosis alone is not subject to compensation.

All workers desirous of entering this industry must, unless they have already completed two hundred hours' work at the time of gazettal of the Act, submit themselves to an "initial examination" by the medical authority appointed under the Act. This examination is made by two clinicians and a radiologist. If at this examination the applicant is found to be suffering from any lung or heart lesion, either past or present, he is refused a certificate and must seek employment elsewhere.

This restriction of choice of an occupation is in the best interests of both the worker and the industry.

A man with damaged lungs should not be exposed to risk from the inhalation of dangerous dust, as, with already damaged lymphatics, he will rapidly accumulate silica and develop fibrosis. From the point of view of the industry which has to support the man when disabled, it is not economically sound to employ any but the healthy.

At this initial examination small pleural scars at the apices are not considered sufficient to bar him, but if such scars are accompanied by glandular enlargement at the hilum, he is refused a certificate, as it is highly probable that he has impaired lymph drainage.

Men with marked catarrhal changes and enlarged bronchial glands are also sometimes excluded, as they are not good "dust" risks.

When a patient presents himself to his regular physician and says he has been refused this certificate, it does not mean that he is seriously ill, but that his future, if he is put to work in silica dust, is not a bright one. Each case is carefully considered on its merits, and the physician must not jump to the conclusion that the medical authority has made a mistake because the result of his examination is "negative".

Men presenting medical certificates to the Committee as suffering from silicosis are frequently found to be suffering from tuberculosis, lung abscess, malignant disease of the lung, or only chronic recurrent bronchitis. Repeated examinations by the medical authority are of importance also in cases of tuberculosis without previous silicosis. It is often found that an apical tuberculosis, as it progresses, assumes a miliary character and is extremely difficult or impossible to distinguish from a silicosis at one examination. Reference to previous examinations will, however, show the progress of the tuberculosis through lungs which at first were quite clear, except at the site of the original tuberculous lesion.

A provision in this Act has added greatly to our knowledge of this disease. This lays down that where dependants claim compensation after the death of a worker who has not been a beneficiary during life, it is necessary to hold a *post mortem* examination. In this way many opportunities are gained of checking up the methods of examination, and it has shown the great accuracy of radiology in the investigation of pneumokoniosis.

The *Workers' Compensation Act, 1926*, allows compensation to workers who become disabled at their work or who are suffering from any disability which might be caused or aggravated by their occupation. This Act covers men suffering from pneumokoniosis used as a general term, but definitely excludes conditions caused by silica dust. As all modern research goes to prove that silica dust is the main damaging factor in pulmonary fibrosis, it can be realized what difficulties arise during an adjudication on claims.

These claims are heard in open court before the *Workers' Compensation Commission*, and it is here that one hears opinions expressed by medical witnesses which are hard to understand and which are contrary to all modern knowledge of this condition.

For one thing, as mentioned above, it is impossible to dissociate a dust fibrosis from silica dust, and the probabilities are that even the pulmonary disabilities of miners are due to the small amount

of silica occurring in the coal. However, coal miners receive compensation under this Act.

There are still many men left outside any compensation act, although they have contracted a silicosis in metalliferous mines; the *Broken Hill Act* applies only to Broken Hill, and the *Silicosis Act* only to the County of Cumberland, and the *Workers' Compensation Act* rules a man out if he has silicosis, and so these men must put up with their disability and manage as best they can.

Radiological Technique.

Before closing, I would refer to technique.

Films should be taken at a distance of four feet or more from the target, and the exposures should be as short as possible and should not be longer than half a second.

Films taken with increasing milliamperages show different appearances, and the same chest taken with 30, 100, 300, 600 and 800 milliamperes shows very different markings, especially when the two latter milliamperages are used.

The detail in a normal lung, using 800 milliamperes at 60 kilovolts, is most bewildering and experience is necessary in interpreting such films. The lung of a young man shows such detail in the lung reticulum with these exposures that most observers would think a pathological condition was present.

I have made several exposures with the rotating target "Metalix" tube at 800 milliamperes and about twenty-five at 600 milliamperes with the 20 kilowatt "Metalix" tube, but the destructive effect on the present day tube prevents these exposures being used as a routine.

I am now using 300 milliamperes for one-thirtieth of a second at seven feet film-target distance, and find the results very satisfactory.

The Potter-Bucky diaphragm should not be used in chest work and all skiagrams should be taken with the patient erect.

Fluoroscopy is of little value in these examinations and is dangerous to the operator.

RENAL PAIN AND NEPHROPEXY WITH OR WITHOUT RESECTION OF THE RENAL NERVES.¹

By M. GRAHAM SUTTON, F.R.C.S. (Edinburgh),
F.R.A.C.S.,

Honorary Assistant Urologist, Brisbane Hospital.

In the first place I wish to thank you for the honour you have done me in having me here tonight and also for the recognition you have given our Post-Graduate Committee in its endeavour to promulgate medical thought and teaching in this State, by inviting medical brethren practising the various specialties to address you. I trust that my efforts will not be disappointing or prove of little practical value.

For a number of years I have been interested in movable kidney, and have given a good deal of thought to the problem, hence the title of my paper, which rather savours of the *multum in parvo*. I hope it is not too much in too little time.

Surgical Anatomy of the Kidney and Ureter.

In order that we may be on common ground I have thought it wise to recall to your minds a few of the salient anatomical facts relating to the kidney and ureter, together with their nerve supply, and then to deal with renal pain, and renal displacement as a cause of renal pain. This will open up the question of acquired renal mobility and displacement, and clear the way to a final discussion on nephropexy.

A line drawn round the body at the level of the umbilicus passes 2.5 centimetres (one inch) below the lower pole of the right kidney, and 3.75 centimetres (one and a half inches) below that of the left. That is to say, the kidneys, together with the spleen, the stomach, and the liver, are supraumbilical organs and are well protected posteriorly and anteriorly by bones and muscles.

Posteriorly, the position of the normal kidney in relation to the last rib is important, in that it furnishes a useful guide to the level at which the kidney should be fixed when operating from the back. The angle formed by the outer edge of the *erector spinae* muscle with the lower border of the last rib is on a line passing from the centre of the hilum to the centre of the convex border of the right kidney.

I would remind you, too, that the last dorsal, the ilio-hypogastric, and the ilio-inguinal nerves pass obliquely behind the kidney and are to be noted and preserved in making and closing the incision; and would point out that, although the pleura is not a direct posterior relation of the kidney, being separated from it by the diaphragm, it is nevertheless important to remember its position when performing nephropexy. Usually the pleura does not extend below the lower border of the last rib, but it may extend lower. So much for the posterior relations.

The anterior relations differ somewhat on the two sides. With regard to the ribs in front, there is a significant observation in respect to their relationship to the kidneys, since they form a bony cage to the upper part of the abdomen, which has been called the middle zone of the body. The lower plane of this zone touches the anterior extremities of the tenth, eleventh, and twelfth ribs, and when lateral or antero-posterior movements are made, or when the person coughs, sneezes, or makes other muscular efforts such as lifting, the lower ribs are adducted, and this plane becomes the point of greatest body constriction.

In persons with normal body form this band of constriction lies below the greater portion of the kidneys, so that, when the body is narrowed by muscular action, the tendency is not to depress these organs, but, on the other hand, to push them up. One may add to this the effect of the upwards and back-

¹ Read at a meeting of the Downs and South Western Medical Association, Queensland, on February 25, 1933.

wards movement of the recti muscles acting on the normally placed colon, and so also pushing up the kidneys.

In persons with other than normal body form it is quite otherwise, and I shall deal with this question later.

The anterior surface of the right kidney is covered on its upper and outer part by peritoneum, which separates it from the liver. Over the hilum and inner border lies the second part of the duodenum; while in contact with the lower part of the anterior surface is the hepatic flexure of the colon. The greater part of the anterior surface of the left kidney is covered by the descending colon.

And this brings me to a consideration of the supports of the kidney itself. The kidneys lie in the deep renal fossæ on either side of the vertebral column. These fossæ are of different shape and size on the two sides and differ materially in capacity and shape in the two sexes, and in persons of different body form in the same sex. Broadly speaking, however, in men they are rather deep depressions that taper downwards resembling an inverted pear. In women of normal body form the fossæ are shallower and more cylindrical, and in some cases wider below, resembling an inverted cone. In both males and females the depth and shape, however, vary with posture and muscular activity. They become deeper and the lower end becomes narrower in the dorsal posture than in the erect posture.

I have already indicated how they are altered in form and capacity by various muscular contractions. The significance of these facts will be more apparent later.

In the renal fossæ the kidneys lie in a special fascial compartment, the arrangement of which has an important bearing on the support of the kidney. This compartment, the perinephric fascia, is separated from the transversalis fascia behind and the peritoneum in front by fat, which is much more abundant posteriorly. The posterior leaf of the fascia is sometimes known as Zukerkandl's fascia and the anterior leaf is known as Gerota's fascia. It is stated that the retrorenal fascia of Zukerkandl is well developed on the left side but very variable on the right. Between the prerenal fascia of Gerota and the peritoneum is the fascial leaf of Toldt which is constant on the left but very limited or absent on the right side.

Now if one follows the peritoneum round the lateral abdominal wall, it will be seen to split into two layers, one of which, the posterior lamella, fuses with the retrorenal fascia, while the other, the anterior lamella, continues forward over the fascia of Gerota. Furthermore, the prerenal and retrorenal fasciæ fuse laterally and above to enclose the kidney and suprarenal body; whereas, below, the fascial sheath is open, except for an inch or so outside the ureter, below the lower pole of the kidney. The prerenal leaf loses itself in the fat of the iliac fossa, while the posterior leaf fuses with the fascia covering the iliopsoas muscle.

These fasciæ by their superior attachments to the diaphragm and their posterior attachments to the vertebrae and the muscles of the posterior abdominal wall afford a very substantial support to the kidney, and it has been claimed a stronger support to the left than the right, by virtue of the anatomical facts just related to you.

But the perinephric fascia encloses a cavity much more spacious than is required to accommodate the kidney which lies within it. Surrounding the kidney is a variable amount of fibro-fatty tissue, its fibro-fatty capsule, or the fatty atmosphere of the kidney. It has generally been supposed that the kidney simply lies in the renal fossa like a bottle packed in straw in a crate lined with tough paper. This conception is, of course, far too mechanical and loses sight of the biological aspect of the question.

Biologically, then, the fibro-fatty tissue acts not only as a "suspensory ligament", so to speak, by its attachment to the perinephric fascia on the one hand, and to the true capsule of the kidney on the other, but also as a lubricating mechanism, which adapts the moving kidney to its fixed surroundings, namely, the perinephric fascia. This can be verified at operation.

With regard to the ureter it has been shown that the relatively more fixed ureter does not move to the same extent as the kidney. It is fixed to the peritoneum on its lower five-sixths, by which its movements are limited. In its upper sixth (four to five centimetres) it leaves the peritoneum and passes through the fibro-fatty perirenal tissue and is mobile, moving with the kidney.

Anatomically the point where the ureter leaves the peritoneum is about the point of crossing of the spermatic or ovarian vessels. This point corresponds to the apex of a triangle formed by the ureter itself on the one side and the spermatic vessels on the other, with the renal vein as a base, and is the highest fixed point of the ureter. The supports of the kidneys, then, in normal persons may be tabulated as follows:

A. General.

- (1) Body form and posture.
- (2) Muscle tone and intraabdominal pressure.
- (3) The perirenal fascia and its attachments.

B. Local.

- (1) The reinforcements to the peritoneum and its attachments to the posterior abdominal wall.
- (2) The blood vessels of the renal pedicle.
- (3) The fibro-fatty tissue which suspends the kidney from the perinephric fascia.

As Kidd succinctly puts it:

The supports of the kidneys in health appear to be intraabdominal pressure, which is maintained by the tone of the unstretched muscles of the abdominal wall in subjects who possess a well formed chest and upper abdomen with sufficient cubic space to hold the heart, the lungs, and the supra-umbilical viscera, and who, in addition, inherit a well balanced and normally functioning nervous system.

The Neuro-Muscular Mechanism of Kidney and Ureter and Renal Pain.

What of the neuro-muscular mechanism of the kidney and ureter and of renal pain? The kidney is

supplied with nerves by way of the renal plexus arising from the semilunar ganglion through which it communicates with the greater and lesser splanchnics, the vagus, and the phrenics, receiving a branch direct from the lesser splanchnic and sometimes one both from the greater splanchnic and the first lumbar ganglion. The splanchnics in turn are formed, mainly by fibres which come from the sixth to the twelfth dorsal nerves, although some fibres are found as high as the fourth dorsal, and some as low as the third or fourth lumbar nerves.

The renal plexus is a network of sympathetic fibres about the vessels of the renal pedicle. The nerve fibres enter the kidney with the renal vessels and follow them to their finest ramifications, small fibres being found on the afferent and efferent vessels to the glomeruli. There are also some fine nerve fibres entering with the small vessels from the fibrofatty capsule, an artery which enters between the suprarenal body and the kidney, and any accessory or polar vessels that may be present. The nerve fibrils in the parenchyma are non-medullated, while those in the pelvis and calyces are medullated.

The nerve supply to the ureter is entirely independent of that to the kidney and comes direct from the lower renal ganglion at the upper end of the spermatic plexus and from the abdominal sympathetics (that is, the aortic, hypogastric, and pelvic plexuses). The nerve supply to the kidney is, by the way, unilateral and as far as the parenchyma is concerned is principally vasomotor with the weight of evidence in favour of a vaso-constrictor influence. It has been proved that section of the splanchnics increases the amount of urine secreted and stimulation lessens it. The vagus does not contain vasomotor fibres.

That there is no secretory nerve to the kidney is universally acknowledged. With regard to sensory nerves, it is apparent that there are sensory efferent nerve fibres to the kidney, which are pathways for reflex influences on renal function. It would seem, therefore, that since vaso-constriction is the main function of the renal nerves, reflex anuria (partial or complete) may be easily caused in this way, possibly aided by spasm of the spiral muscles of the renal papillæ which are probably supplied by the same nerves.

Coming now to afferent sensory impulses, there is no doubt that renal pain comes by way of sympathetic fibres, but that these stimuli are opposed except when very strong. This view is held by Müller, Head, and MacKenzie, who concede that painful sensations from the sympathetic area in general are felt only when they reach the *rami communicantes* of the posterior spinal nerve roots and the cerebro-spinal centres. The result is, that pain is referred to the area of distribution of those nerves in which the corresponding *rami communicantes* end. These areas are known as the zones of hyperæsthesia of Head.

In regard to the kidneys, this area is that supplied by the tenth, eleventh and twelfth dorsal nerves and the first lumbar nerve.

Concerning the nerve supply of the tissues surrounding the kidney you will remember that Frazer, of Edinburgh, has found it useful for clinical purposes to divide the abdominal parietal peritoneum into demonstrative and non-demonstrative areas.

The demonstrative area is the larger and includes all the lining of the abdominal cavity except the pelvis and that part of the posterior abdominal wall bounded roughly by the ascending and descending colon and the transverse meso-colon. This latter is the non-demonstrative area. The non-demonstrative area is probably supplied chiefly by the sympathetic system; the demonstrative by the main somatic nerves. In the renal regions there are more nerves in the tissues behind the kidneys than in the peritoneum in front of them, so that the latter regions should be included in the non-demonstrative area.

In a practical way, then, the clinical application of these facts relating to the nerve supply of the kidney, ureter, and surrounding tissues depends upon the nature of the irritant and the various ways in which the irritation of the nerve terminals is made known to us. Such irritation may be evinced clinically by: (i) local pain and tenderness, (ii) referred pain, (iii) superficial hyperæsthesia and hyperalgesia, (iv) muscle rigidity, (v) alteration of muscle reflexes.

The characteristics of renal and ureteral pain may be stated briefly to be of two types, capsular and pelvic.

Symptomatically capsular pain is recognized as a dull lumbar ache; pelvic pain as the sharp renal colic radiating round the loin to the groin. But with all pelvic pain there is probably an associated capsular element, for pelvic distension produces capsular stretching. On the other hand, abnormality in or about the capsule may cause pain unassociated with any disturbance of the pelvis or ureter. The pain felt at the beginning of renal colic is localized at a point where the anterior axillary line crosses the costal arch. It is a referred pain in one of Head's zones of hyperæsthesia familiar to you all. And while on the subject of referred pain I would like to relate the following history of a patient who complained of shoulder pain.

CASE I. B.J.W., a male, aged twenty-nine, married, a baker, complained of an ache in the left groin ever since he was in Brisbane Hospital two years ago next June, when I removed a grossly tuberculous right kidney after several examinations to exclude tubercle bacilli from the left kidney and to ascertain its function. Also he had tuberculous prostatitis. He now complains of pain over the left trapezius and supraspinatus fossa coming down over front of clavicle and chest, also a burning pain in left loin. He had nausea, but never vomited when the pain came on. He had scalding on micturition, but not "like before". He passed urine three times during the day and not at night. His appetite was good. His tongue was clean. He had gained weight. He had no cough. There was no pain on respiration. X ray examination of the chest revealed no abnormality.

Cystoscopy revealed a healthy bladder. Dye in the function test appeared on the left side in seven minutes.

The colour was good. Urine from the left side was clear; no pus was present but a few red cells were seen.

Pyelogram taken on January 11, 1933, revealed a hydro-nephrosis with a caterpillar bend in the ureter.

This patient's shoulder pain is interesting, since it was this he came about and it was only after questioning him that he mentioned the other pain; and of course, I knew his history.

Regarding phrenic shoulder pain, Z. Cope in his book, "Clinical Researches in Acute Abdominal Disease", writes:

Some years ago Mr. Mayo Robson wrote an interesting note on three cases in which pain at the tip of the shoulder was a prominent symptom and in none of these was it caused by gall-stones.

All three were due to tumours growing at the upper end of the kidney. The explanation offered for the pain was that a small branch of the phrenic passes to the semilunar ganglion. In view of the necessary irritation of the tissues over the diaphragm by such tumours, that explanation is hardly needed.

Pelvic pain is usually accompanied by reflex phenomena such as nausea, vomiting, pallor, cold sweats, but without any change in pulse or respiration.

On palpation a healthy kidney lying in normal position is not sensitive. If, however, a mobile kidney is grasped between the fingers there is often a sensation of pain, like that experienced when the testicle is squeezed in a similar manner. I have noticed that when one has grasped and held such a kidney and then lets it slip up under the hypochondrium, the patient jumps and indicates pain. This I believe is due to the hypersensitiveness of the perirenal fibro-fatty tissue.

It is stated that incision of the kidney, that is, nephrotomy, under local anaesthesia is painless, but that palpation of the pelvis itself or traction on the pedicle and separation of the peritoneum is at once complained of.

Movable Kidney.

It will now be profitable to discuss the problem of movable kidney; how it is caused, and how it produces symptoms.

The condition occurs after puberty, and is seldom found in children. Virtually it is a disease of women in adult life, principally in the third and fourth decades. It is often associated with general splanchnoptosis in which the abdominal viscera are abnormally mobile. This type of case is in a class by itself and need not detain us longer.

I have already indicated the normal supports of the kidney, but let me reiterate so as to crystallize in your minds the mechanism by which the position of the kidney is governed, first, from a retentive point of view, and secondly, from a dislocative or disruptive point of view, for, as I hope to show you, movable kidney is in reality a rupture, a hernia, an internal hernia.

A. The retentive factors are of two types, dynamic and static.

(1) The retentive dynamic forces are:

(a) The tone of the muscles of the abdominal wall, which presupposes a well balanced neuro-muscular reflex arc, that is, normal distribution of intra-abdominal pressure.

(b) The elasticity and resiliency of the fibres and strands of the healthy fibro-fatty tissue and the adequacy of the perinephric fascia, living tissue, not straw and paper.

(c) The pull of the renal pedicle on the great vessels, a bow.

(d) The motility of the anterior upper abdominal wall in an ambulatory individual, not a mummy.

(2) The retentive static factors are:

(a) The anatomical configuration of the mid-zone of the human body, that is, the shape and cubic capacity of the upper part of the abdomen and chest in the well developed adult.

(b) The attachments of the peritoneum and the ligaments of the supraumbilical viscera, including those of the perinephric fascia itself.

B. The disruptive factors are also of the same two types, dynamic and static.

(1) The disruptive dynamic forces are:

(a) Sudden acute or repeated chronic traumata, for example, blows and falls; railroad, car and horse riding, accidents *et cetera*.

(b) Oft-repeated small thrusts to an organ at a mechanical disadvantage (by reason of the static disruptive factors below), for example, chronic cough.

(2) The disruptive static factors are:

(a) The upright position in man.

(b) The loss of cubic space in the mid-zone of the body, for example, the tendency of the barrel shaped chest of infancy to become long and narrow at adolescence, the widening of the pelvis in females at puberty and the congenital flat chest type of body form.

(c) The loss of muscle tone and tissue vitality in the perinephric fibro-fatty tissue.

The retentive and the disruptive factors in the normal state, relative to the mid-zone of the body, must balance; if they do not, acquired renal displacement occurs.

Broadly speaking, the three most important events in the aetiology of movable kidney are diminution in cubic space, loss of muscle tone and thrust.

The dynamic disruptive forces are always forthcoming if the static disruptive factors eventuate. It is like the second stage of labour; when the stage is set, the expulsive forces prevail.

But how do these factors operate in practice? In a series of patients with movable kidney there will be found two types of individual. Firstly there is the well developed woman with a broad well formed chest of the natural adult type, the antero-posterior diameter very little less than the lateral, and with a wide subcostal arch. She may have a slight stoop and rounded shoulders with a protuberant abdomen and as likely as not a placid disposition. Owing to

the large size of the renal fossæ in broad chested persons the "suspensory ligaments" of the kidney are not so liable to rupture with sudden trauma or so subject to over-stretching with repeated traumas, presumably because the kidney is free to move more antero-posteriorly and does not necessarily receive the downward thrust as it would in a more confined space.

When these patients have movable kidney, they suffer usually from a more or less general splanchnoptosis and possibly also from pelvic prolapse and really are suffering from *hernia en glissant* of the supraumbilical viscera in general, from the plane of greatest body constriction and of the kidney only incidentally.

Certainly the kidneys have herniated from the renal fossæ, but the ligamentous complications that produce pain and call for operation do not arise as a rule and they are to be treated by exercises, stimulation of the abdominal muscles, and later perhaps provided with a belt.

Secondly, there is the type of patient with the narrow flat chest and narrow subcostal angle and narrow abdomen with good abdominal muscles and often with an ill-balanced and poorly functioning nervous system. This peculiarity of body form may be congenital or acquired, but it is essentially a female type (although many of the patients have narrow hips) and only a few men conform to it.

These patients may be recognized also by their excitable nature and erotic temperament, and are often pretty and *petite*, but are easily tired and exhausted by the trials and tribulations of life, and have a lowered threshold for pain. Owing to their type of body form they have insufficient room in their chest for their heart and lungs and as a consequence their diaphragm and pleuræ are placed too low and their supraumbilical organs tend to lie at a low level compared with the broad chested person. The muscles of the abdominal wall are strong and taut, but they have small renal fossæ and their kidneys are too movable, because, being in a relatively confined space, either at or just below "the plane of greatest body constriction", the kidney is subject to disruptive dynamic factors acting at an advantage. The conclusions of Frank Kidd are so convincing that I can not do better than quote him. He writes:

It would appear, then, that a kidney can become too movable, if the abdominal muscles become weak or stretched, or if the chest from inheritance or at adolescence becomes too long, flat or narrow, so that the heart, lungs, diaphragm, and supraumbilical viscera are pressed downwards towards the pelvis.

In either case the restrainers of movement, such as the vessels of the pedicle and the so-called "ligament of the kidney", must become stretched and lengthened, and, at the same time, an attempt is made on the part of the fibroblasts to thicken the fibro-fatty perinephric tissue.

This fibrosis occurs especially round the lower pole of the kidney and the upper end of the ureter and may become so marked as to cause painful adhesions and to fix the lower pole and prevent it from moving upwards, and also fixes the kink in the ureter.

Pain, therefore, may be caused by the pull on the nerves of the renal pedicle, sprain of "the ligament", the development of adhesions, or by kinking of the ureter at its highest fixed point, with consequent intermittent blocking of the outflow of urine from the kidney pelvis with resultant early hydronephrosis.

The increased mobility and intermittent blocking of the ureter predispose the kidney to attacks of bacterial inflammation—i.e., pyelitis—which may be another cause of pain.

Such inflammation, which fixes the ureter to a greater extent than the kidney, tends to fix the kink in the ureter, and so aggravates the trouble. In these ways movable kidney becomes a diseased state.

We can now recapitulate by saying:

1. That movable kidney occurs in women because they belong to the sex which is liable to inherit or acquire a long narrow chest and because they are liable to lax abdominal walls.

2. That it occurs chiefly on the right side because the diaphragm on the right side has a greater thrust through the liver, a heavy organ; because the colon on the right side drops much more easily than the left, which is more effectively anchored; and lastly because the peritoneum is reinforced on the left side, and possibly also the renal niche on the left side has a better retentive shape.

3. That sudden trauma can either rupture or stretch the upper portion of the fibrous bands or "ligaments of the kidney" which support it, from the perinephric fascia—a fixed structure—or it can rupture the prerenal fascia or its line of fusion below the lower pole of the kidney. That is to say, thrust is overwhelming even in a person not otherwise predisposed, causing torn ligament *et cetera* and pain.

4. That, quite apart from acute trauma, the chronic trauma caused by a low diaphragm in those persons with narrow chests can stretch these suspensory ligaments and gradually herniate the prerenal fascia or its line of fusion below the lower pole of the kidney. That is to say, thrust is at an advantage in certain predisposed persons, causing lax ligaments *et cetera* and movable kidney.

5. That although in most women with the narrow type of chest and abdomen the right kidney can be felt to be movable, the majority of them do not complain of pain, either because their nervous systems are, as yet, in a state of balance, or their ligaments have not been subject to sudden trauma or to super-added inflammation and they have not yet developed painful adhesions.

6. That by reason of certain complications a too movable kidney may cause pain and become a diseased state.

Symptoms and Signs.

Patients with movable kidney may complain simply of a renal drag which is relieved by rest and aggravated by standing. This is due to the adhesions formed and is really a condition of sprain comparable to sprain of other ligaments in the body, for example, in joints.

They may complain of attacks of typical renal colic accompanied by oliguria. This is due to kinking of the ureter (strangulation) and possibly in some

cases to torsion of the pedicle (Dietl's crises) comparable to the symptoms of transient or partial strangulation in other herniae.

They may complain of attacks of pyelitis and fever. Faulty drainage or stasis and congestion due to the mobility and adhesions explains this infection (which is commonly due to the *Bacillus coli communis*) and its tendency to persist.

They may show hæmaturia; and in young adults orthostatic or intermittent albuminuria has been ascribed to the congestion produced by stress on the renal vessels.

They may complain of vomiting and dyspepsia and even of jaundice, though I have never seen a case in which jaundice was present. These symptoms are supposed to be due to traction of the right kidney on the second part of the duodenum and the bile ducts, and if so, they will be relieved by rest on the left side with the foot of the bed raised, or by abdominal support.

They may suffer from neurasthenia, partly primary and partly secondary to the renal sympathetic stimulation and/or the pain.

Diagnosis.

The diagnosis, then, is made by the history, abdominal palpation, the exclusion of other abdominal lesions, a flat X ray picture, but chiefly by pyelography. We are not able to do this with the patient in the upright and the Trendelenburg positions, which perhaps is the ideal, on account of the difficulty with the Potter-Bucky diaphragm, but we compromise by using the dorsal position at expiration and the 45° position at inspiration, when possible.

Every patient with a movable kidney who has renal symptoms is given a full urological examination. A flat X ray examination is made to exclude stone. Cystoscopy is undertaken to perform the dye-function test and to obtain catheter specimens of urine to ascertain whether there is infection or not, and to rule out obstruction in the lower part of the ureter. A pyelogram is necessary to demonstrate kinks of the ureter and hydronephrosis, and may be used as a guide to the emptying time of the renal pelvis by exposing a film eight to ten minutes later, after having drawn the catheter into the lower part of the ureter.

Normally the renal pelvis should be empty in ten minutes at the outside. If infection is present and especially if it is unilateral and on the side of the kidney that has undergone ptosis, it is added evidence and demonstrates the need for nephropexy. Sometimes renal lavage may be carried out if the economic and social circumstances of the patient make it necessary to postpone operation. I must be convinced that the pain is definitely of the renal type, and I look with doubt on the diagnosis if pain is not relieved by rest, and if it is not aggravated when the patient is up and about. It must be conceded, however, that the renal ache due to perirenal fibrosis may quite well come on at night, much in

the same way as in other painful adhesions after any kind of sprain.

Also, I lay great stress on the presence of a kink in the upper part of the ureter, especially if it is accompanied by a dilatation of the pelvis and blunting of the calyces with widening of their necks. Of course I realize that this dilatation may not be entirely mechanical, but may be partly due to a reflex or inflammatory loss of tone of the musculature. In the absence of hydronephrosis, infection is a deciding factor in advising nephropexy. Here let me relate a relevant case history.

E.J., a female, aged fifty-one, married, had a dragging pain in the right loin for eight months. Then she had a severe right renal colic which sent her to bed, and she required morphine to obtain relief. Pyuria and palpable kidney were discovered by her doctor, who gave her citrate of potash and diluents and advised her to wear a belt. This relieved the lumbar ache.

Two weeks previous to my seeing her she had a second attack of colic and had a flat X ray examination made which showed the kidney shadow to be low. She was then referred to me for investigation.

Cystoscopy revealed a healthy bladder. The dye-function test yielded dye from the left side in three minutes, and from the right side in five minutes. The colour was good. Urine from the bladder contained pus cells and motile bacilli. The right kidney urine contained abundant red cells, some pus and epithelial cells.

A pyelogram showed a large pelvis and calyces with clubbing. This was more an inflammatory relaxation than a hydronephrotic dilatation as was indicated by their blurred outline. There was a negative shadow in the pelvis; it was thought that this might be due to a uric acid stone. The ureter was lax; ptosis of kidney was present, but no kink.

Operation was performed on December 5, 1930. A low mobile kidney was found and easily brought up. Dissection disclosed a double pelvis and explained the apparent negative shadow. Pyelotomy revealed no stone. A number 9 F. ureter bougie was passed down the ureter. Then a number 7 E. red rubber catheter with two holes cut in it was passed through a nephrostomy opening into the upper calyx and brought out above the twelfth rib. Nephropexy was performed. The catheter drained well. The patient has remained well and free from pain, her doctor says.

Lastly, I am always more convinced when, at pyelography, on filling the pelvis the patient volunteers the identity of the pain produced with that she previously complained of, or admits it afterwards. But, of course, this applies only to those patients who complain of the colicky type of pain; and I would point out also, that some patients with fairly well advanced hydronephrosis experience no pain, even when the pelvis is completely filled.

My decision to operate, then, is based on my conviction that certain patients with movable kidney are relieved of their symptoms by operation. But of course, not all patients with movable kidney are troubled by them, and others who are, are quite unsuitable for operation.

I exclude straight away as unsuitable, firstly, patients with general splanchnoptosis; secondly, patients with chronic cough (just as I believe that old men with chronic cough or prostatic obstruction should not be operated on for hernia or should have their prostates enucleated first, as the case may be); thirdly, patients who have become confirmed neurasthenics.

Suitable patients are those who have periods of brightness and contentment and who show a desire or even an anxiety to be well; who suffer intermittently from renal ache or renal drag, which is relieved by recumbency; who suffer from Dietl's crises or from superadded attacks of pyelitis; who give a definite history of sudden onset after a severe trauma, or who are numbered among the phthinoid group. Most of the patients selected should conform to this type with good abdominal muscles.

The Operation of Nephropexy.

Nothing has done more to bring discredit on the operation of nephropexy than the fact that it has so often been performed in unsuitable cases, in a haphazard way, without proper attention to detail. I have already told you how I pick my cases.

With regard to the operation itself, there are several points which require emphasis, so I will briefly outline the technique which I follow, namely, that of Frank Kidd.

The patient is anaesthetized in the dorsal position and then turned on her side with the elbow tucked under her body, well back, and with the top leg straight and the under leg flexed at the hip and knee joints. Besides this, it is most essential to have the kidney bar or sandbag in place, so as to open up the space between the last rib and the iliac crest. Neglect of this point will occasion the loss of much time and cause much difficulty later, when the kidney is delivered.

The incision that I prefer is either a simple vertical one made just outside the outer border of the *erector spinæ* muscles, or an angular one. The upper limb of the angular incision commences well above the last rib and extends downwards vertically just outside the *erector spinæ* muscles for about three inches; the lower limb of the incision runs forward from this 2.5 centimetres (an inch) or so above the iliac crest for some 5.0 centimetres (two inches).

The incision is deepened until the last rib is exposed in the upper part of the incision and the lumbar fascia in the lower. Now is a good time to dissect the *erector spinæ* up from the twelfth rib so as to expose the external arcuate ligament. All bleeding points should now be tied, and there will be a number of veins requiring ligation in the upper angle of the wound.

Next, the lumbar fascia is opened, and at this stage a sharp look-out must be kept for the twelfth dorsal, the ilio-hypogastric and ilio-inguinal nerves, which lie just beneath this layer and which are particularly liable to injury.

If branches of the accompanying blood vessels are cut, care must be taken in applying forceps and ligatures so as not to bruise these nerves or include them in ligatures. Injuries to these nerves leads to a band of anaesthesia just below the iliac crest and a band of hyperaesthesia along the area supplied by the eleventh dorsal nerve. This painful area is later apt to be taken for the pain of appendicitis, especially if the patient is seen by another surgeon.

Not only this, but nerve injury is said to lead to weakness of the muscles in the iliac region, and even to inguinal hernia.

The perinephric fascia should next be stripped off the *quadratus lumborum* muscle, as it is better and easier to do this at this stage than after the kidney has been delivered *et cetera*.

This being done, the perinephric fascia is opened well back so as to avoid opening the peritoneum, though no harm is done if it is opened. As a matter of fact, I have removed the appendix in this way from the lower angle of the wound and also have removed the gall-bladder in a patient with gall-stones and movable kidney. It gives a particularly fine exposure to the gall-bladder, as the intestines fall away and cause no trouble. Some surgeons advocate opening the peritoneum as a routine so as to make the operation an exploratory one. The perinephric fascia having been opened, the kidney is stripped out from its bed and delivered on to the loin. This is most easily accomplished by hooking the finger round the ligament at the upper pole and drawing the kidney out, upper end first. With the kidney on the loin, all adhesions and fat are carefully separated, preferably by scissors curved on the flat. Each adhesion is examined before being divided, so as to avoid accessory vessels, since these retract deeply into the loin and may cause troublesome bleeding. One is struck by the toughness of some of these adhesions, especially at the lower pole.

Careful dissection is now carried out over the pelvis and posterior surface of the pedicle so as to leave them bare. This step, partially at any rate, denerves the kidney and renders it less sensitive to causes of renal pain. The ureter is freed well down below the iliac crest and any abnormal set of blood vessels running behind or in front of it is looked for and the vessels are divided between ligatures.

Having freed the ureter and made sure there are no obstructing bands or aberrant vessels, we are now ready to decapsulate the upper half of the kidney and insert the suspension sutures.

A small nick is made in the capsule of the kidney near the upper pole on the outer border. Through this a dural elevator is inserted and the capsule is separated. It is then incised all round the outer border and folded back so as to leave the upper half of the kidney quite bare. The bleeding is seldom troublesome and soon stops if the kidney is wrapped in hot sponges or pads of gauze. Through the portion of the capsule which is turned down like a collar, two fine chromicized gut stitches are passed, each in the form of an inverted "U", taking up small bights of the capsule only, on the back of the kidney. I deprecate the passage of sutures into or through the renal parenchyma as unnecessary and harmful.

The ends are left long, and each in turn is carried up from before backwards through the posterior abdominal wall. The anterior pair is passed by means of a hernia needle above the twelfth rib, care being taken to push aside the pleura (with a

finger of the left hand), which sometimes bulges down in this location through a hiatus in the diaphragm. The posterior pair is passed through the external arcuate ligament in similar manner.

The kidney is now unwrapped and replaced in the renal fossa, and as it is pushed up under the twelfth rib and the diaphragm, the two pairs of stitches are drawn taut, not tight, and tied mattress fashion.

The kidney will now be seen to be well tucked up, with only its lower pole showing, and will be seen to be fixed, that is, not to move with respiration—an entirely unnatural state of affairs.

What happens is this: in the course of a few weeks fresh adhesions form between the bared upper half of the kidney and the posterior abdominal wall, and the stitches gradually cut themselves loose.

The movements of respiration gradually lengthen the newly formed adhesions and the kidney once more becomes a movable organ, but is still anchored in its new position above the plane of greatest body constriction, and is less subject to the displacing or dislocating forces, while the ureter has been straightened out and runs direct to the bony pelvis unfettered at any one point more than another.

The kidney bar is lowered or the sandbag is removed and the wound is closed in layers with meticulous care; for lumbar hernia is not uncommon, and just as much misery can be caused by the inclusion of the nerves in sutures as by cutting or injuring them whilst making the incision. It is wise to leave a glove drain in the lower angle of the wound for three to four days to allow the escape of blood or sero-fatty fluid from around the lower part of the renal fossa.

The patient is returned to bed on her back and nursed thus for three weeks, with the foot of the bed raised on blocks for the first ten days.

When the operation is performed in this way in suitable cases, I have not had any reason to doubt the efficacy of the procedure either from a technical or a therapeutic point of view.

I have had occasion two or three times to take a pyelogram subsequent to such operations, and in each case the kidney has remained where I put it.

It may be of interest to add that this operation may be extended to include resection of the renal nerves for renal pain of uncertain aetiology, such as occurs in painful nephritides, small, painful, non-obstructive hydronephrosis, and a condition called "the painful abnormal motility syndrome" (Harry Harris).

All one has been able to offer these patients in the past is renal decapsulation, temporary nephrostomy or nephropexy. These have usually proved insufficient, while nephrectomy is far too drastic. There is no more difficult problem than that of the small, painful hydronephrosis. In such a patient we can only offer nephrectomy of an almost normal kidney.

In this operation, namely, renal sympathectomy (Ambard and Papin), after exposure of the kidney

the renal pedicle is wiped bare with gauze and the nerve trunks are picked up with tissue forceps and severed across a grooved director with a curved tip. It is not difficult to distinguish the resistant nerves from the soft lymphatics. Both aspects of the pedicle are dealt with in turn. One ought to pass between the vessels to do the job thoroughly, and it is very tedious; injury to the renal vein can easily occur, as it did in one of my cases. After the denervation one completes the operation with a nephropexy.

The post-operative pain is worse, I think, than after ordinary kidney operations, but otherwise there is no difference.

I have done this operation as such in five cases with complete failure in one, partial relief in two, and relief of pain in the other two, so that I am not yet convinced of its efficacy.

Case Histories.

I shall now conclude by showing the pyelograms of several of my cases of nephropexy, with pertinent remarks on their history and the results obtained. This will be facilitated, first, by showing you a drawing of the anatomical outline of the normal kidney pelvis on the screen, followed by two or three pyelograms of normally placed kidneys; secondly, by a more or less arbitrary clinical division of the thirty cases I have collected, in which both the full history and pyelograms have been traceable.

The clinical subdivision is as follows: (i) Cases illustrating the effect of trauma as an aetiological factor, two cases. (ii) Cases of nephroptosis without infection, four cases. (iii) Cases of ptosis with infection, that is, pyelitis, nine cases. (iv) Cases of ptosis with hydronephrosis, three cases. (v) Cases calling for discrimination in diagnosis and treatment, five cases. (vi) Cases of nephralgia or "painful abnormal motility syndrome", that is, cases in which it is difficult to explain renal pain on the basis of ptosis, infection or obstruction, seven cases.

The accompanying table shows the cases classified into these six groups.

The following are short accounts of cases typical of these groups.

The following two cases illustrate the effect of trauma.

G.B., a male, aged thirty-four years, a caterpillar tractor driver, complained of pain in the right loin at the back coming round to the front. He had nausea, but no vomiting. Sometimes he had frequency of micturition. He thought there was some relation between the pain in the right loin and the bladder irritability. He never had any hematuria. He had an accident three to four years ago. He was crushed between buffers, three ribs were broken on the right side and the collarbone was lifted at the inner end. He dates his trouble from this. Cranking the big engine brings on the pain.

The right kidney is palpable. A flat X ray examination reveals no abnormality. A pyelogram was made on October 28, 1929. This gave him the same pain, but more severe, and made him vomit. Nephroptosis was present. Nephropexy was advised, but he postponed it. This is a case illustrating effect of severe trauma.

Table showing Clinical Classification of Cases of Nephroptosis and Nephralgia.

(I) Cases illustrating trauma as an aetiological factor.

Number.	Initials.	Age.	Sex.	Parity.	Diagnosis.	Treatment.	Remarks.
1	G.B.	34	M.	—	Nephroptosis.	Nephropexy advised.	Patient postponed operation.
2	A.M.	38	M.	—	Nephroptosis.	Nephropexy.	Good result.

(II) Cases of nephroptosis without infection.

3	G.L.H.	27	F.	3	Ptois and kink.	Nephropexy.	Good result.
4	E.A.H.	32	M.	—	Ptois, angulation at pelvis.	Plastic on ureter. Nephropexy.	Relieved.
5	G.D.	28	F.	Nil.	Ptois, angulation.	Nephropexy.	Good result.
6	V.K.	28	F.	1	Ptois.	Nephropexy.	Good result.

(III) Cases of nephroptosis with infection.

7	M.C.	?	F.	?	Right pyelitis, ptois. Pregnant.	Medical.	—
8	M.McW.	28	F.	Nil.	Pyelitis, ptois.	Medical.	Cleared up.
9	J.A.T.	28	F.	2	Left pyelitis, kinked ureter.	Nephrolysis and nephropexy.	Relieved for two years.
10	E.C.	25	F.	Nil.	Left pyelitis, ptois.	Medical.	Cleared up.
11	E.J.	51	F.	?	Right pyelitis, ptois.	Nephrotomy and nephropexy.	Good result.
12	E.S.C.	48	F.	?	Left pyelitis, right ptois. Cystitis.	Medical and bladder lavage.	Not relieved.
13	M.G.	24	F.	Nil.	Double pyelitis and ptois.	Double nephropexy.	Good result.
14	M.McB.	26	F.	3	Left pyelitis, ptois, kinked ureter.	Medical.	Too recent.
15	D.W.	21	F.	Nil.	Pyelitis, ptois, kinked ureter.	Nephropexy.	Too recent.

(IV) Cases with hydronephrosis.

16	C.F.	32	F.	Nil.	Strictured meatus, hydronephrosis.	Referred to own doctor.	—
17	E.J.K.	40	F.	?	Infected hydronephrosis.	Nephrolysis and nephropexy.	Good result.
18	B.J.W.	29	M.	—	Kinked ureter and hydronephrosis.	Not operated on yet.	—

(V) Cases for discrimination.

19	N.R.	22	F.	Nil.	Nephroptosis and nephralgia.	Operation not advised.	—
20	S.P.S.	25	F.	Nil.	Congenital ectopia.	Operation not advised.	—
21	L.V.	17	F.	Nil.	Stricture of right ureter, left pyelitis.	Dilatation of ureter, medical.	Relieved.
22	L.M.F.	50	F.	3	Gall stones, nephroptosis.	Cholecystectomy, nephropexy.	Good result.
23	V.M.	22	F.	—	Early nephroptosis.	Abdominal exercise, belt.	Too recent.

(VI) Cases of nephralgia or painful abnormal motility syndrome.

24	A.T.	42	F.	Nil.	Nephralgia.	Renal sympathectomy, nephropexy.	Failure.
25	M.B.	35	F.	3	Tortuous ureter.	Renal sympathectomy, nephropexy.	Partial relief.
26	C.R.O.	49	F.	4	P.A.M.S., double kidney.	Renal sympathectomy, nephropexy.	Relieved.
27	G.	30	F.	1	Small painful hydronephrosis.	Renal sympathectomy, nephropexy.	Relieved.
28	E.S.	27	F.	Nil.	P.A.M.S.	Renal sympathectomy, nephropexy.	Partial relief.
29	V.D.	36	F.	Nil.	? P.A.M.S., ? pyelitis.	Medical. Eserine.	—
30	F.McG.	19	F.	Nil.	? P.A.M.S.	Medical. Eserine.	—

A.M., a male, aged thirty-eight years, a labourer, was well until three months ago and then had sudden onset of pain which shot down to the right testis; he felt nauseated, but had no vomiting, no scalding or frequency, and the urine was not smoky. Since then he had had a constant ache in the right loin, intensified by work. He had twelve attacks of the severe pain. The right kidney was palpable and tender. There were no previous illnesses or operations.

Cystoscopy revealed a normal bladder. The dye-function test revealed an excretion time of seven minutes for both sides; the colour was good. In the urine no pus cells, *et cetera*, were seen. Culture was sterile. A pyelogram showed the kidney to be in good position, but the patient was horizontal. There was a kink in the ureter opposite the fourth lumbar vertebra.

Operation was performed on October 5, 1931. Nephrolysis and nephropexy were done. On discharge on November 5,

1931, the patient stated that the pain in the loin had gone, but that pain was present in the right iliac fossa on extending the right thigh. This seems to have been due to some tacking down of the ureter to the psoas muscle, and it finally disappeared. On January 8, 1932, he reported free from all pain.

The following case is one of nephroptosis without infection.

G.D., a female, aged 28 years, single, complained of pain in the left renal area on and off for five years. At Christmas, 1931, she had frequency of micturition and dysuria and pain in the back on the left side. She had no hæmaturia. She was referred to me on February 25, 1932, for a urological survey.

Cystoscopy revealed a normal bladder. Both ureteric meati were congenitally narrow. The left was catheterized

easily to the pelvis. The dye-function test revealed an excretion time of five minutes on both sides. The bladder urine was clear. No pus cells were present. In the left ureter urine there were no pus cells or organisms, but a few red cells and a trace of albumin. A pyelogram revealed a large pelvis, with good cupping. There was ptosis of the kidney, with angulation and a deviated ureter.

Nephrolysis and nephropexy were performed, and the patient is definitely relieved of the pain to date.

The following is an example of ptosis with infection.

M.G., a female, aged twenty-four, single, complained of severe renal colic on the right side coming on suddenly at work. She had frequency of micturition and pyuria.

The right pyelogram showed acquired ptosis with a kinked ureter. Right nephrolysis and nephropexy were performed by me on June 21, 1932. A few days after returning home the patient got severe left sided colic, rigor, vomiting, frequency of micturition, pyrexia *et cetera*.

Cystoscopy revealed mild cystitis. In urine from the left ureter there were a few pus cells and staphylococci. The pyelogram showed a condition on the left similar to that on the right. Left nephropexy was performed on August 26, 1932.

After being treated for the pyelitis medically and the cystitis topically, she has eventually regained health, lost her pain and frequency of micturition and put on weight.

The following case is an example of ptosis with hydronephrosis.

E.J.K., a female, aged forty years, married, complained of pain in the right loin radiating to the groin for twelve months. She had some frequency of micturition, but no hæmaturia. The pain was worse on movement and following a day's work.

In the bladder urine a few pus cells, a number of epithelial cells and motile bacilli were present. The right ureter urine contained a number of pus, red and epithelial cells; no casts were seen. Motile bacilli were present.

A pyelogram revealed ptosis, hydronephrosis which was infected, and a kinked ureter.

At operation on April 17, 1931, the kidney was found to be very mobile. Nephrolysis was difficult. Nephropexy was performed. The patient was apparently relieved.

The following is an example of the class of case calling for discrimination in diagnosis and treatment.

L.V., a female, aged seventeen years, single, complained three weeks ago of pain in the right iliac fossa and nausea. She had no vomiting and no rigor. She had had previous similar attacks on two or three occasions. No definite frequency of micturition was present.

A catheter specimen of urine contained a number of pus cells, a few red and epithelial cells. A flat X ray examination revealed no calculus. Cystoscopy revealed a healthy bladder. Dye-function test gave satisfactory results. The right kidney urine contained a few epithelial cells but no organisms. The left kidney urine contained a few pus cells and a number of red and epithelial cells.

A pyelogram taken with the patient in an inclined position showed the kidney to be low. Blunting of the calyces with widening of their necks was present. There was a suggestion of stricture at the pelvic brim. No kink was present.

After the pyelogram was taken the patient stated that she had not had the pain in the lumbar region before, but the pain lower down was similar to the pain she complained of.

The diagnosis on July 15, 1932, thus seemed to be left mild pyelitis, with a probable stricture of the right ureter at the pelvic brim. Operation was not advised and the patient was treated by dilatation of the right ureter with a number 11 F. ureteral bougie twice. Citrate of potash and later hexamine were given. According to the notes she was free from pain on September 23 and October 21, 1932.

The following case is typical of the last group, that of neuralgia or the painful, abnormal motility syndrome.

G., a female, aged thirty years, married, with one child, complained of pain in the right loin for a considerable time. She had had her appendix removed and had undergone operation for retroversion of the uterus. She had had various forms of medical treatment, but still had the aching pain in the loin.

Her urological investigation notes have been mislaid, but her pyelogram, November 23, 1927, shows a fairly well placed kidney, but with evidence of early hydronephrotic clubbing of the calyces and a bifid pelvis.

I performed renal denervation and I believe she was relieved, as her husband paid the account on the time payment system and always stated she was better.

Acknowledgements.

Finally, I wish to acknowledge my indebtedness to the teachings of Mathé, Frank Kidd, and also of Kelly and Burnam, who have done so much towards the elucidation of the problems herein discussed and who have so materially aided in placing the treatment of movable kidney on a rational basis.

THE TREATMENT OF CARCINOMA OF THE UTERINE CERVIX.¹

By ROBERT FOWLER, M.D., F.R.C.S.,

Honorary Gynaecologist, Alfred Hospital, Melbourne;
Honorary Gynaecologist, Austin Hospital,
Melbourne.

In September, 1931, THE MEDICAL JOURNAL OF AUSTRALIA published a communication by myself upon the treatment of uterine cancer. The present article may be regarded as a protocol of this publication, embodying as it does the revision of our results and experience to May, 1933.

A record is available of every patient with cervical carcinoma who has sought our advice during the last nine years (Dr. J. M. Buchanan acted as my deputy during 1932). These records number 177 and their analysis represents the sum total of my individual experience in private and institutional practice. The circumstances of practice, however, have been such that 74 of these patients were committed to decisive therapy by other clinicians prior to coming under the care of our service. Deducting these, there remains a series of 103 in whom we were primarily responsible for the determination of deliberate therapy. Of these, five refused to submit to treatment, and in seventeen the disease was so far advanced that even radiological treatment was withheld.

Summarizing the foregoing, we obtain the following statement:

Of 177 patients seen in our service during the last nine years, 81 were patients treated deliberately, 17 were patients too advanced for treatment. This is a total of 98 for whom we accept complete responsibility (Category A).

Five were patients who refused treatment, 74 were patients who were treated for recurrence *et cetera* after previous treatment elsewhere. This is

¹ Read in abstract at the Fourth Australian Cancer Conference, Canberra, March, 1933.

a total of 79 for whom we accept only partial responsibility (Category B).

With this grouping of the statistical material we may briefly study Category B (Table I) and thenceforward concern ourselves entirely with Category A (Tables II, III, IV and V).

The melancholy tale told by Table I is intelligible only if it is realized that most patients in Category B are seen at the Austin Hospital for Incurables, the inevitable "dumping ground" for therapeutic failures. They are transferred to our service for supplementary treatment of residual growth, recurrence or complications.

TABLE I.
Carcinoma Uterine Cervix.
Analysis showing fate of 79 cases in Category B.

Detail.	Total.	Alive.	Dead.	Untraced.
Patients refusing treatment ..	5	—	1	4
Patients treated for recurrence of <i>cervix</i> after previous treatment elsewhere ..	74	3	64	7
Calculated to May, 1933 ..	79	3 ¹	65 ¹	11

¹These three survivals are attributable to radio-therapy initially instituted over five years ago by Dr. A. H. Thwaites (two cases) and Dr. J. L. Clendinning (one case).

²The duration of life following treatment in these cases has not yet been worked out. All the patients died of cancer; one was known to have survived seven years after initial radiological treatment.

An analysis of the significant features in the treatment of patients in Category A may now be followed through the succeeding tables (II, III, IV and V).

Our methods of treatment have been detailed in the article already referred to (*THE MEDICAL JOURNAL OF AUSTRALIA*, September 5, 1931). They are also indicated in Table II. "Excision" signifies the radical abdominal operation of Wertheim in all save three cases (one a resection of cervix, one an extended vaginal excision, the other a two-stage abdomino-perineal operation). "Radium" signifies the local application of radium element, chiefly by the Paris technique. No supplementary irradiation has been used, except in a few instances (external X radiation; transperitoneal radon insemination).

"Combined" signifies the preliminary application of radium followed at various intervals from four weeks to forty weeks by abdominal excision.

TABLE II.
Carcinoma Uterine Cervix.
Analysis of 98 cases (Category A) to show Applicability of Treatment.

Date.	Decisive Treatment.				Case Total.	Percentage of Total Treated.
	Ex-cision.	Radium.	Com-bined.	Nil.		
Prior to—						
1926 ..	4	0	0	1	5	
1926 ..	2	1	1	2	6	
1927 ..	3	0	1	2	6	
1928 ..	8	3	1	2	14	
1929 ..	2	11	0	1	14	
1930 ..	0	16	2	2	20	
1931 ..	1	10	2	3	15	
1932 ..	1	9	3	5	18	
Totals ..	21	50	10	17	98	83%

(a) 21 of the 50 "radium cases" were "operable"; add the 31 operated upon—52 "operable" out of 98 (53%).

(b) 31 of these cases (Category A) occurred over five years ago. 24 of these were suitable for treatment; seven were untreated. Six of these are still alive (five out of 20 operated on; one out of four radiated). 19%—absolute cure rate. 25%—relative cure rate.

It will be noticed that our predilection for surgical treatment in the earlier years has given place since 1928 to a preference for radium therapy. It is noteworthy that coincident with this change our case totals have markedly increased. This is interpreted to mean that popular apprehension of a stupendous surgical operation deters patients from seeking advice, whereas the prospect of radiotherapy has not nearly so marked an effect. If, as appears likely, this deduction is true, it must be reckoned as one of the most telling advantages of radiotherapy.

The statistical sample analysed in Table II contains 17 cases of such advanced disease that not even palliative radiotherapy was considered advisable. This circumstance resulted in a relatively low tractability rate (83%). It must be emphasized that our standard of tractability is an elastic one, depending upon the varying severity of the cases and upon whether the prospect is one of cure or

TABLE III.
Carcinoma Uterine Cervix (Stages 1 and 2).
Comparison of Surgical, Radium and Combined Treatment in 52 Cases.

Character of Treatment.	Detail.	1924.	1925.	1926.	1927.	1928.	1929.	1930.	1931.	1932.	Results over—		
											Total Period.	Three Years Ago.	Five Years Ago.
Excision alone	Total ..	2	2	2	3	8	2	—	1	1	21	19	17
	Alive (1933) ..	—	—	1	—	4	2	—	—	1	8	7	5
	Percentage Survival ..	—	—	—	—	—	—	—	—	—	38%	37%	30%
Radium followed by excision.	Total ..	—	—	1	1	1	—	2	2	3	10	5	3
	Alive (1933) ..	—	—	—	—	—	—	2	0	3	5	2	0
	Percentage Survival ..	—	—	—	—	—	—	—	—	—	50%	40%	0%
Radium alone ("operable" cases only).	Total ..	—	—	—	—	2	5	6	4	4	21	13	2
	Alive (1933) ..	—	—	—	—	1	5	3	1	3	13	9	1
	Percentage Survival ..	—	—	—	—	—	—	—	—	—	62%	69%	50%

TABLE IV.
Carcinoma Uterine Cervix.
Analysis of 31 Patients on whom Surgical Excision was Practised.

Duration since Operation.	Date of Operation.	Total.	Alive.	Dead.	Remarks.
Years.					
9	1924	2		2	
8	1925	2		2	
7	1926	2	1	1	Combined abdomino-perineal operation. Pre-operative radium.
		1		1	
6	1927	1		1	Pre-operative radium. Operation by Victor Bonney.
5	1928	8	4	4	One of the patients alive had extended vaginal excision.
4	1929	2	2	1	Pre-operative radium. One of the patients alive had reaction of the cervix only.
3	1930	2	2	1	Pre-operative radium.
2	1931	1		1	Pre-operative radium.
1	1932	2	1	1	Pre-operative radium.
		2	2	1	Pre-operative radium.
Totals		31	13	18	Calculated to May, 1933.

NOTE.—The patient who in 1929 had an apparently inadequate operation and who now appears cured, suffered from an unusually circumscribed carcinoma microscopically proven.

merely palliation. For example, in Table V are shown all patients who have had radium applications, irrespective of the object in view (detergent, curative, palliative) or the stage of the disease. Of our tractable cases, 52 would have been classified according to surgical standards as "operable" (operability rate, 53%).

It is in this group that deliberate therapy, whatever its nature, aims at a cure. First rate surgeons have shown that approximately two out of five may be cured by surgical operation alone; the same result has been attained by front rank radio-therapists. In our management of this group we have striven to imitate both schools; our results are shown in Tables III and IV.

Table III presents results of treatment by different methods. These results are comparable, since

TABLE V.
Carcinoma Uterine Cervix.
Analysis of 60 Patients on whom Initially Radium Therapy was Practised.

Duration since Treatment.	Year of Treatment.	Total. ¹			Stage 1.			Stage 2.			Stage 3.			Stage 4.			Character of Treatment.
		A.	D.	U.	A.	D.	U.	A.	D.	U.	A.	D.	U.	A.	D.	U.	
Years.																	
7	1926		1								1						Radium only.
			1														Radium and excision.
6	1927		1					1									Radium and excision.
5	1928	1	2					1	1		1						Radium only.
			1						1								Radium and excision.
4	1929	6	5		2			3			1	5					Radium only.
3	1930	4	12		1			2	3			9		1			Radium only.
		2	2					2	1		2	2		1	1		Radium and excision.
2	1931	2	6	2		1		1	2		2	2					Radium only.
		2	2					2	2								Radium and excision.
1	1932	4	4	1	2			1	1		1	3				1	Radium only.
		2	1					2	1								Radium and excision.
Totals		21	36	3	5	1		12	13		2	21	2	2	1	1	
Totals distinguishing operable from in-operable		60			31 "operable."			29 "inoperable."									

¹Calculated to May, 1933. A—Alive. D—Dead. U—Untraced.

only "operable" cases are considered. By "operable" is meant "early" and "borderline" cases, or Stages 1 and 2 of Döderlein's classification (League of Nations). There is no need for "hair-splitting" over the definition of "operability" by those of us brought up in similar surgical schools. I am sure that any Australian surgeon, who has performed upwards of ten or a dozen radical excisions for cervical cancer, would classify the operability of our cases in a manner similar to our own or differing by not more than 2%. In any case, the significance of "operability" is out of date. The use of surgical operation alone for cervical carcinoma is fast becoming obsolete. This statement, I am aware, would be challenged by Bonney (London), Wagner (Berlin), Weibel (Vienna), and Faure (Paris), but is amply warranted by the tenor of the discussion at the British Medical Association centenary meeting (London, 1932) and by observations I have made recently at important centres in Europe and America.

An increasing body of opinion supports the thesis that decisive therapy in cervical carcinoma should be entirely radiological—a combination of cavitation radium application with distance irradiation by high voltage X rays or a radium "bomb".

Among those adhering to this view may be mentioned Forsell and Heyman (Stockholm), Regaud and Lacassagne (Paris), Döderlein and Voltz (Münich), Hurdon and Donaldson (London), Grey Ward (New York), Cutler (Chicago), Bowing (Mayo Clinic), Moran (Sydney).

Table III indicates that our results with radium alone are going to be better than those attained by surgery alone, but if experience abroad is any guide, these results would be still better had we utilized auxiliary external irradiation. In future we hope to make this a constant feature of our technique except in very early cases.

From time to time numerous gynaecological surgeons, including ourselves, have exploited various combinations of radium therapy and surgical

excision. A favourite method is to apply radium to the cervical growth a few weeks prior to radical extirpation by either the abdominal or vaginal route. It should be noted that this method depends for its success upon the completeness of the operation, the radium being utilized merely for its detergent action. Our limited experience of this plan (utilizing the abdominal route) has not been favourable, although in two instances where radium failed and excision was undertaken after seven and ten months respectively, we entertain a reasonable expectation of cure.

We have performed but one extensive vaginal excision for operable collum-carcinoma. Stoekel (Berlin) and Adler (Vienna) always practise this method combined with radium therapy. The former applies radium before operation, the latter immediately afterwards.

In my previous article in this journal I postulated five advantages of radium therapy in comparison with surgery for cervical carcinoma. I should like to review these in the light of further experience.

"It does not deter patients from seeking advice." The probable truth of this postulate has been sustained by our experience, as already discussed in this paper.

"It is applicable to almost all cases presenting." As already indicated, we do not apply radium in every instance of cervical carcinoma. Although it is never too early for such treatment, it may occasionally be too late. With radium available we have treated 83% of our patients beneficially, whereas had we to rely solely upon surgical means, our tractability rate would have been no more than 53%.

"It is devoid of primary mortality and mutilation." This postulate is not absolutely true. In common with others, my series shows a small primary mortality. Two of these patients (one a diabetic) died of peritonitis following cavital radium treatment.

"Its technique is easier of acquisition than is operative skill." I still believe this to be true, although it is a fact that, whereas most gynaecologists are trained in surgery, few are trained in radiology. This is a pity, because I am convinced that the proper person to control the treatment of uterine cancer is the gynaecologist, and that a modern service for the treatment of gynaecological cancer should provide facilities for both surgical and radiological treatment. It is to be hoped that the diplomas of radiology recently instituted in the Universities of Sydney and Melbourne will prove popular.

"Its results are rapidly improving." Perhaps we may now drop the word "rapidly" from this postulate. Our results in Australia are definitely improving and will continue to do so in proportion to the improvement in our facilities for auxiliary external irradiation. We are in need of higher voltage X ray equipment and of a radium "bomb". Moreover, we require to utilize much more freely the services of the physicist in calibrating and standardizing dosages *et cetera*. Both these matters

might well engage the attention of public cancer committees forthwith.

Notwithstanding every facility, it seems that radiotherapy, like surgery, has reached the limit of its capacity to cure the average case of *carcinoma colli*. Such a state of affairs is no reflection upon the value of these methods, since the average case is no true test of merit. Given thoroughly early cases of strictly localized disease, then 85% to 90% of cures may be expected from either surgery or radiation. Moreover, if surgery were to be the method of choice, a much less drastic excision might replace the operation of Wertheim or Shauta.

Unfortunately, the detection of incipient collum-carcinoma is infrequent; despite prolific propaganda and conscientious clinical conduct, the average patient reaches the treatment centre with the disease well established. The reason is to be found in the insidiousness of the change from pre-cancer to cancer, and it is doubtful if we shall solve the problem of early diagnosis until more is known of aetiology. Nevertheless, it is probable that much may be gained by the more general use of vital staining with Lugol's solution (Schiller), colposcopic examination (Hinselmann), and serological tests (Bendien), as well as scrupulous attention to the so-called pre-cancerous cervix.

In conclusion, one may be permitted again to stress the importance of organization in the control of cancer.

Clinical endeavour forms but a part of the concerted social effort. The necessary cooperation of pathological and physical laboratories is fairly well appreciated, but sedulous "follow-up" of treated patients is not as generally practised as it should be. In this direction clinicians sadly need a helping hand from statisticians and social welfare workers. Apart from the reproach attaching to failure in detecting recurrence of the disease at the earliest possible moment, an inefficient "follow-up" system involves us in a further difficulty. It precludes a precise statement of the extent of our ability to cure visceral cancer. This achievement may be certainly attributed to both surgery and irradiation, although unimpeachable figures would make an irresistible public appeal.

So much for the more technical aspects of the problem, to say nothing of such matters as propaganda and finance.

The integration of each and all of the several factors involved in cancer control is obviously a function of some central health authority. It is remarkable, and, I think, a matter for regret, that the State of Victoria is the only one in the Commonwealth that has not yet organized a public anti-cancer campaign.

Summary.

1. A statistical report is presented concerning the progress of a personal series of cases treated for carcinoma of the uterine cervix.

2. A commentary on various practical points is included.

3. Criticism is offered concerning certain features of cancer control organization and equipment in Australia, together with suggestions for improvement.

Reports of Cases.

VARICELLA AND HERPES ZOSTER.

By G. A. MURRAY, M.B., D.P.H.,
Medical Officer, Commonwealth Department
of Health, Sydney.

SINCE 1892, when von Bokag, of Budapest, suggested that it might be possible for the virus of *herpes zoster* to cause varicella in another person, much evidence has been gradually accumulating in support of his suggestion, and it is considered that the following case is sufficiently suggestive of such relationship to place on record.

A passenger vessel trading to Japan and China was granted full pratique at Melbourne on April 24 after having been fully inspected by medical officers of the Commonwealth Department of Health at Brisbane, Sydney and Melbourne. Three days later, on April 27, F.S., a cabin boy employed in the first class accommodation, developed *herpes zoster*. Later, four cases of varicella occurred on board, one fourteen days, two fifteen days, and one eighteen days from the date on which the herpes case commenced.

These cases were:

H.Y., first class Chinese bath boy. Rash appeared on May 10.

T.M., first class Chinese cabin boy. Rash appeared on May 11.

C.K., first class Chinese lamp boy. Rash appeared on May 14.

D.W., child, European, second class passenger. Rash appeared on May 11.

H.Y. and T.M. occupied the same sleeping quarters as F.S., and contact was practically continuous. C.K., whilst not occupying the same sleeping quarters, occupied an adjoining cabin, and contact with the other three Chinese was regular and frequent. T.M. and C.K. had been on shore at several ports, but H.Y. had not been ashore for twenty-nine days prior to the onset of his illness.

Full examination was made of all the stewards on the occurrence of these cases, to detect any further cases or any person showing signs of a recent attack of varicella. No such person was detected.

As it was considered possible that F.S. was the source of infection in cases H.Y. and T.M., he was selected to accompany them to the Quarantine Station to assist in cooking and service during their detention. Though in close contact with these throughout he did not develop varicella. He states that he has never had varicella.

D.W. boarded the vessel on May 2 at Melbourne. He is stated to have had no contact with any case of varicella on shore. He felt sick on May 9 and the rash appeared on May 11. No contact with the three Chinese could be established and, since their duties were confined to the first class accommodation, contact with a child in the second class would not be likely, whilst the period of incubation would be unusually short if his infection had occurred on board. It would appear probable that this patient was infected on shore prior to joining the vessel.

The rather isolated and self-contained nature of life among Asiatic crews of overseas vessels presents advantages in studying such an outbreak, as opportunities for outside infection are limited and the system of repeated medical examination up to the eighteenth day from the last overseas port of call affords definite knowledge of the conditions on board.

In the vessel now under consideration the dates of onset and the close association of the Chinese contracting varicella strongly suggest a common source of infection. Since T.M. had not been ashore at any port, and H.Y. and C.K. had not been ashore together, if the suggestion of a common source of infection be accepted, this must have

been on the vessel. The Chinese, closely questioned, state that they had no visitors in common at any port. If this is accepted (there appears no reason for untruthfulness in this connexion), the infection must have arisen from a member of the ship's company. From the repeated medical inspections of all on board—inspections designed to detect such infectious conditions—it may be accepted that no previous case of varicella had occurred on board during the voyage. Therefore it is considered that the probabilities are in favour of the case of *herpes zoster* being the source of infection in the Chinese patients. No significant features were discovered which might be regarded as antecedent to this case of *herpes zoster*.

Reviews.

THE MEDICO-LEGAL ASPECT OF WORKERS' COMPENSATION.

THE compensation of injured workers is steadily assuming more importance to the medical profession, and Sir John Collie's "Workmen's Compensation: Its Medical Aspect" is a welcome addition to the rather meagre medical literature on the subject.¹ Sir John Collie admits to thirty years' experience in the working of the English *Workmen's Compensation Act*, and the legal references in his book relate to that Act, which provides compensation for "personal injury by accident" and certain diseases which are limited to those set out in a schedule to the Act.

The underlying principles of the English Act in general are similar to those of the Australian States, and the principles of law laid down by the English Appellate Courts are followed where applicable by the tribunals administering these laws in Australia. Many of these principles are clearly set out in this book, but readers must first inform themselves of the provisions of the Act operating in their own State. The New South Wales *Workers' Compensation Act, 1926-1929*, for example, does not require a worker to prove that the personal injury he sustains was "by accident" although, of course, it must not be intentionally self-inflicted, and compensable diseases are not scheduled as in the English Act, but all diseases (other than those caused by silica) gradually contracted or otherwise due to the nature of the worker's employment, which arise out of and in the course of his employment, are compensable by the New South Wales employer, who is also compelled by the statute to indemnify injured workers for cost of medical and hospital treatment and to insure his liability with a licensed insurer. (In New South Wales there is special legislation for diseases caused by silica.)

The responsibilities of the medical examiner are fully discussed in the book and call for even greater care, in certain Australian States at least, than in England, as in many cases where the injured worker has a dependent wife and several children, there is no monetary incentive for him to return to work, the compensation he receives for himself, wife and children equalling his average weekly earnings where they are less than £5.

A careful perusal is recommended of the chapters on the law and medical examination; the law with regard to operations; the position on complete or partial recovery; return to work; *novus actus interveniens*; medical referees; and the relation of rheumatism and fibrosis to accidents, which will prove helpful to many medical practitioners. Chapter XVII, relating to the giving of evidence, is particularly valuable. The New South Wales *Workers' Compensation Commission*, when medical questions are at issue, always avails itself of the right to have a medical assessor sit with it, a practice which is not universally adopted in England, nor in the other States of Australia.

We endorse Sir John Collie's opinion that: "A medical man's functions under these Acts are somewhat analogous to those of a chartered accountant under others: he is intended to be the unbiased professional man who shall adjust the balance evenly between conflicting interests."

¹ "Workmen's Compensation: Its Medical Aspect", by Sir John Collie, C.M.G., D.L., M.D.; 1933. London: Edward Arnold and Company. Demy 8vo., pp. 160. Price: 7s. 6d. net.

The Medical Journal of Australia

SATURDAY, JULY 29, 1933.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE PRACTITIONER AND HIS JOURNALS.

MEDICAL journals are of various types and are published with diverse objects. Certain journals, the organs perhaps of special societies or of research institutes, are used to record the results of investigations undertaken by society or institute; these journals are often highly technical and are appreciated probably by relatively few. Other journals, while dealing with research, have a certain clinical bearing and are sought by those who wish to gain complete understanding of the scientific basis of a specialty and who aim at perfecting themselves in its art. Again, there is a type of journal intended more especially for general practitioners. In journals of this type are published articles dealing with everyday subjects; sometimes a *résumé* of knowledge on an ordinary subject is given, sometimes a subject of everyday interest is presented in a new light. In journals of this type also certain authors attempt to correlate the results of research with clinical experience, and others record clinical observations which can be used by laboratory workers and those whose daily labours do not take

them to the bedside. Of course, all journals cannot be put into one of these three categories, nor is it desirable that they should belong rigidly to one type. It is good for every kind of worker that he should sometimes leave his own domain to investigate others. Particularly are these excursions necessary to the mental health of the clinical worker. Most clinicians find this out for themselves sooner or later, when they are faced with the need for finding out all that is to be known of a subject. It follows that medical practitioners, be they specialists or in general practice, must have access to medical journals of every type.

In Australia, severed as it is from the well equipped libraries in the old world, medical practitioners often find themselves, as the Israelites of old, oppressed by Pharaoh in Egypt, found themselves, compelled to look for straw to make bricks. Like the Israelites, they are sometimes forced to use stubble instead of straw. If they make inferior bricks, they will not have the same justification as had the oppressed people in Egypt. They can get their straw if they would have it.

On previous occasions the library problem of Australian practitioners has been discussed in these pages and suggestions have been made for the pooling of resources in large centres; the avoidance of overlapping has also been emphasized. At first sight it might appear that the methods adopted in the larger capitals would not be applicable in the smaller cities. This view is not correct; the same principles can be applied. There is no need to repeat the arguments previously used, but since every avenue must be explored, reference will be made to the provision of journals by groups of practitioners and by societies. Groups of practitioners sometimes meet in study circles. Members of these groups subscribe to certain journals; if the journal for subscription has been chosen wisely, the copies might with advantage be donated eventually to the nearest medical library. Some of the Australian Branches of the British Medical Association have sections for the study of special branches of medical science. The subscription to these sections is small, and the whole of the subscription is not required for the running of the affairs of the section. In

some States the sections have determined to donate to the Branch library one or more journals. This system should be extended to all the Branches. Again, in many of the States there are medical societies, not affiliated with the British Medical Association. These organizations also collect a small annual subscription which is allowed to accumulate. In certain instances quite a large sum has been gathered. These societies might consider the advisability of presenting to the library of the Branch of the British Medical Association or to some other library in the State an endowment, the interest of which should be used as a subscription to special journals which are not at present available in Australia. In all efforts to enlarge the library facilities of Australian practitioners, however, care must be taken to prevent duplication of subscription, particularly to any of the more expensive foreign journals.

Current Comment.

ADHESIONS IN THE UPPER PART OF THE ABDOMEN.

It is well known that peritoneal adhesions are not uncommon in the abdomen particularly in the right upper quadrant. As important organs are located here, it is of great interest to know just what bearing these adhesions may have upon visceral function. The gall-bladder and duodenum are not only organs subject to considerable functional stress, but they are not uncommonly the site of inflammatory disease. Moreover, they produce very definite direct and reflex symptoms when so affected. Therefore, the clinician naturally turns to the hypothesis of adhesions when no other demonstrable cause for his patient's upper abdominal discomfort can be found. Still more naturally the surgeon, seeing this abnormal state under his eyes in the open abdomen, seizes on the same explanation. Whether or not this is warranted by fact or reasonable deduction is worth serious thought. We know that the abdominal viscera may be well harnessed in adhesions without gross symptoms being detected, but this is no proof that in other instances such symptoms may not arise.

An inquiry into this question has been made by S. G. Meyers and A. R. Bloom, who have studied the subject from the combined standpoints of the anatomist, the clinician and the radiologist.¹ As a preliminary they review the literature, and from this it is readily apparent that upper abdominal adhesions occur frequently. This agreement

amongst various authorities fails to be sustained when we come to the important matters of the cause and the effect.

The method of study adopted by Meyers and Bloom is to combine autopsy information with clinical investigation and radiological evidence obtained by simultaneous filling of the gall-bladder and duodenum. They report a large percentage of congenital membranes related to the colon, duodenum and gall-bladder. They point out that the gastro-hepatic omentum may extend to the neck of the gall-bladder and to the first and second portions of the duodenum, or even may invest the fundus of the gall-bladder and pass to the transverse colon below. These anomalies must not be mistaken for inflammatory adhesions. The radiological study showed that the combined method adopted was valuable in demonstrating deformities of the organs in question, but Meyers and Bloom point out that the findings must not be unduly stressed. For example, a normal gall-bladder may leave a "seat" on the duodenum, and a diseased gall-bladder may fail so to do: therefore this "deformity" of the duodenum, though suggestive of an inflamed gall-bladder, is not proof of such a lesion. From the clinical standpoint the authors examined patients in an endeavour to find whether deformity of the gall-bladder indicated the presence of adhesions, and whether deformity of the duodenum occurred particularly in the presence of known gall-bladder disease or other morbid state. In only one-half of their cases with a radiologically deformed gall-bladder were there any pathological changes demonstrable in the right upper abdominal quadrant, and in only one-third of such cases was there clinical evidence of cholecystitis. In regard to the duodenum much the same was found. Ulcer accounted for 40% of the radiological deformities, cholecystic disease 25%. In the remainder, over 30%, no clinical entity could be built up to fit the X ray findings. In cases of proved gall-bladder disease only about 20% showed a shifting or definite deformity of the duodenal cap. The Graham test was here much more useful than the opaque meal.

The conclusion reached is that membranous adhesions occur so frequently in the right upper abdominal quadrant that great caution is needed before it is justifiable to assume that they are inflammatory in cause or harmful in effect. The combined method of radiological study yielded comparatively little positive information as to the presence or effect of such adhesions. Thus it seems as if here again we have evidence that the demonstration of an anatomical aberration from standard, whether congenital or acquired, does not warrant the assumption of a pathological state. Of course there must be many persons who are happily unconscious of being the possessors of "abnormal" bands and membranes in their upper abdominal quadrants. Further; the knowledge could be of no service to them unless a definite medical or surgical purpose was thereby to be served. But most difficult of all is the problem of the patient who still suffers

¹ *Archives of Internal Medicine*, April, 1933.

discomfort after an abdominal operation and who is known to have adhesions of the inflammatory order. Confirmation of his possession of a lesion for which the remedy is unsure cannot reassure him; attempts to belittle the cause of his troubles will often make him more dissatisfied and introspective. The authors point out that Clairmont and Meyer, of Zurich, in a communication on this subject considered that 62% of their patients had a psychic defect and advised prudence in attaching clinical significance to radiological deviations from the accepted standard. In view of this depressing statement it would seem all the more wise to insure that all persons who have needed extended medical or surgical treatment for an upper abdominal lesion should be thought of as persons and not merely as the external casing of an errant organ. Too many people resume their work and face the cares of life again all too soon after illness and operation, and a realization of this truth should do much for the future of all those luckless enough to be laid low by any disabling ailment.

PROTEIN DIGESTION AND ACHLORHYDRIA.

THE introduction and wide adoption of the fractional test meal has not only stimulated interest in the chemistry of the gastric secretion in those suffering from digestive disorders; it has revealed secretory anomalies and defects in persons who are apparently healthy. Of these anomalies the absence of hydrochloric acid is one of the most interesting. Some of the persons discovered with this defect appear quite normal in other respects. Some are suffering from other debilitating or toxic diseases, and the question is sometimes raised whether the secretory defect is causal or merely a result of other systemic disease. Another interesting group is the familial, including relatives of those suffering from pernicious anaemia. In all these persons it is of interest to speculate as to the harmful effect, if any, that might arise through an anacid gastric juice.

The normal antiseptic power of the secretion is impaired, and alimentary tract infection may ensue. Again, there may be failure to produce a necessary hemopoietic factor, and a macrocytic type of anaemia may result. But apart from these possibilities, how does the achlorhydric person carry out the digestive function, particularly as regards protein?

This problem is attacked by D. C. Hines in a recent communication.¹ The patients studied all showed complete anacidity in the test meal, even after the administration of histamine. The method adopted was the comparison of the faecal nitrogen during periods of high and of low nitrogen ingestion. Assuming that digested protein was absorbed, the excreted nitrogen would be a measure of completeness of protein digestion. The low nitrogen diet comprised vegetables, fruit, fat and carbohydrate, the caloric value being 1,900, with a

daily nitrogen content of less than 1.5 grammes. The high nitrogen diet contained enough meat to bring the nitrogen value up to 20 grammes, calories and bulk being unaltered. Fluids were kept constant. The diets were given for three-day periods and the total nitrogen was determined in urine and faeces corresponding to these periods. No change could be demonstrated in nitrogen excretion, and this remained virtually constant despite the considerable alterations in the nitrogen of the diet. Hence it was assumed that the achlorhydric person was quite competent to digest protein. This result has been obtained by previous workers in the same field. The value of these confirmatory findings lies in the fact that the patients had a persistent and apparently true anacidity, for only a few cubic centimetres of neutral mucus could be obtained even after injecting histamine.

Further experimentation was done to find if the rate of digestion was altered in these patients. Equilibrium was insured by a three to five day period, during which a standard diet was given containing 1.2 grammes of protein per kilogram body weight. After an overnight fast the patient was encouraged to eat as much beef steak as possible. No other food was given until evening, when a 1,000 calorie supper with less than one gramme of protein was allowed. The meat was in some instances ground before being cooked, but eaten normally; in others it was cooked whole and swallowed in small cubes without chewing. The rates of digestion were then measured by curves constructed from serial analyses of the nitrogen excreted. Normal controls showed results closely agreeing with those of former investigators. When the urea excretion rate in the urine was used as an indicator, no difference in digestion rate appeared to occur whether the finely divided meat was bolted or swallowed after mastication. But in two patients diarrhoea occurred after the unchewed meat was swallowed, and in these there was naturally a large loss of nitrogen in the stools. Provided that diarrhoea did not occur, the achlorhydric patients showed no variation from the normal in their rate of protein digestion as measured by this method. The conclusion arrived at is that digestion of protein in the subjects of gastric anacidity is disturbed neither in degree nor in rate. As the author remarks, this explains why such persons can retain a high grade of nutrition over long periods.

It is comforting to have further assurance that these persons can carry out their proper digestive work efficiently. Of course, it may be advisable to give hydrochloric acid if there are other indications, such as the occurrence of pernicious anaemia in other members of the family, or a possible peroral infection, or gastrogenous diarrhoea. However, this work stresses the need for fine division of the food in the manner designed by Nature. No digestive juice, adequate or inadequate, can cope with a massive and undivided bolus of food, and the old advice to eat slowly and masticate well still holds good.

¹ *The American Journal of the Medical Sciences*, May, 1933.

Abstracts from Current Medical Literature.

SURGERY.

Diffuse Polyposis of the Stomach.

NEAL SINCLAIR (*The British Journal of Surgery*, April, 1933) describes the eighty-fifth case on record of diffuse polyposis of the stomach. A female, aged fifty-seven years, experienced post-prandial epigastric pain for fourteen years; vomiting and diarrhoea had been present for six months. The use of alcohol had been very moderate. X ray examination revealed well marked hour-glass deformity of the stomach. At operation a thickened stomach wall was found, and a partial gastrectomy (including the distal loculus) was performed. Eight months after operation the patient was in good health. The specimen removed measured 12.5 centimetres along the lesser curvature. The diameter of constriction was 18 millimetres. Dark coloured polypoid masses projected from the surface, resembling a number of prolapsed and oedematous piles. There was no evidence of ulceration. The aetiology of the disease is obscure. A congenital factor may be present, for the condition has been found in twins and in mother and son. The author compares this with the familial tendency in the analogous condition of the small intestine and colon. The condition bears many names. The tumours are generally referred to as adenomata. In many cases there has been evidence of chronic inflammation of the gastric mucosa. Papillary adenomata have been produced experimentally by the injection of coal tar into the submucosa of rabbits' stomachs. The author thinks that the sequence of events in his recorded case was probably as follows. The patient had a gastric ulcer which healed, producing a bilocular stomach. This was associated with an unusual degree of chronic gastritis in the distal loculus. This led to thickening of mucosa and formation of inflammatory polypi, leading to progressive obstruction of the pylorus with increasing stasis and further gastritis; thus a vicious circle was established. Carcinoma has not been found so frequently with polyposis of the stomach as it has been with polyposis of the colon. There are no characteristic symptoms of the disease, which in most instances has been diagnosed at operation or autopsy. In those cases in which test meals have been carried out, achlorhydria was a constant finding.

Syphilis of the Stomach.

K. A. MEYER AND H. A. SINGER (*Archives of Surgery*, March, 1933) discuss syphilis of the stomach and make particular reference to its recognition by the surgeon at operation. They point out that a frozen section made of tissue removed for biopsy during operation is of relatively little aid in establishing a diagnosis of gastric syphilis. The value of the

microscopic preparation, provided the block of tissue is properly chosen, lies chiefly in the exclusion of carcinoma. The diagnosis can best be made by a consideration of the gross characteristics of the lesion. There is a striking disparity in many cases between the extent of the lesion as determined by X ray examination and the extent as observed at operation by palpation. Whereas in carcinoma one generally finds at laparotomy a more extensive involvement than the X ray picture indicates, in syphilis of the stomach there is a striking paucity or complete absence of changes as determined by palpation. The common lesion of gastric syphilis is not a spherical prominence, as in a tumour, gummatous or neoplastic, but is a flat infiltrate that leads to a plaque-like thickening of the gastric wall. When thin, the infiltrate is readily overlooked. When it is thick, its relatively soft consistency and pliability distinguish it from carcinoma. On cross-section, the increase is seen to be due to an oedematous fibrous tissue located chiefly in the submucosa. Inspection of the interior of the stomach generally discloses one or more superficial, serpiginous ulcers involving a large part of the plateau formed by the infiltrate. The base of the syphilitic ulcer, unlike the peptic variety, is situated at a higher level than the normal mucous membrane. Fibrous and oedematous thickening of the serosa and adhesions, when present, suggest an inflammatory rather than a neoplastic lesion. Associated syphilitic changes that may be detected during the course of an operation, include hepatic, intestinal and splenic syphilis. Of these, the coexistence in the liver of gumata, deep stellate scars, or subtotal destruction of the left lobe, is of the greatest aid in the recognition of the type of gastric disease present.

The Effect of Atropine on the Secretion of Transplanted Gastric Pouches.

EUGENE KLEIN (*Archives of Surgery*, February, 1933) writes on the effect of atropine on the secretion of transplanted gastric pouches. In the dog, atropine in non-toxic doses (1.0 to 1.5 milligrammes) can abolish the secretion of hydrochloric acid produced by food in the Pavlov pouch, in the Heidenhain pouch, and in transplanted subcutaneous gastric pouches without the myenteric plexus. In the last mentioned type the parasympathetic (vagi) and the pre-ganglionic and post-ganglionic sympathetic nerves are eliminated. Whether the post-ganglionic parasympathetic nerves are also absent cannot be stated until the relations of the submucous plexus are definitely known. At any rate, following food, only stimuli through the blood could reach this pouch. These stimuli could be completely eliminated by one milligramme of atropine. The method by which the humoral secretion from food is inhibited by atropine in the transplanted pouches is not clear.

This action may take place either in the antrum or peripherally at the cells. One milligramme of atropine does not abolish the secretion of one cubic centimetre of histamine in these pouches. The histamine probably acts directly on the cell. The same dose of atropine that could abolish stimulation by food when given fifteen minutes before a meal was ineffectual when given after a meal. This fact may be of use clinically. In one dog with a transplanted pouch a spontaneous secretion was present at irregular intervals not associated with the test meal. This secretion was diminished by one milligramme of atropine, but was not abolished. When it was present and a test meal was given, the atropine did not abolish the secretion. This spontaneous secretion could not have been due to vagus stimulation. It is possible that it was of parenteral origin and that its action was directly on the cell. In man atropine apparently does not abolish the chemical phase of secretion. The differences and possible causes are briefly discussed. It is possible that variations in the proportionate amount of the primary (cephalic) and secondary (chemical) phases of secretion partly account for the lower acidity in gastric ulcers as compared with duodenal, and the far higher percentage of anacidity following partial gastrectomy for gastric ulcer. If in gastric ulcer a larger proportion of the secretion is due to the chemical phase from the antrum, the removal of this part of the stomach would account for the greater frequency of the anacidity.

Osteomyelitis of the Skull.

ALFRED W. ADSON (*The Western Journal of Surgery, Obstetrics and Gynecology*, February, 1933) discusses the surgical treatment of osteomyelitis of the skull. The treatment does not differ from the treatment of osteomyelitis of other bones; removal of all necrotic and infected osseous tissue is required in addition to sequestrectomy. However, special consideration concerning these operative procedures is necessary, since the soft tissues, such as the scalp and periosteum, may have been destroyed, and removal of infected bony tissue may result in exposure of the meninges. A lesion needs further special consideration, since brain abscesses are frequent sequelae of osteomyelitis of the skull. Adequate drainage is necessary, all necrotic and white dead bone should be removed until live, bleeding bone is exposed, both outer and inner tables should be removed if diseased, and the dura exposed if necessary, regardless of the presence or absence of scalp covering the area. The wound should be cleansed by removing all infected soft tissue, and antiseptic solutions should be employed to prevent or retard bacterial growth, radical measures, with wide exploration, should be advocated in preference to drainage by stab wound, in order to prevent progress of the infection. Regeneration of bone

to repair defects in the skull depends on the periosteum and osseous tissue. The skull readily regenerates from either of the tables, and when one table has been removed, the defect is soon closed by the osseous proliferation. When both tables of the skull have been removed, regeneration can occur from the periosteum, or it may take place from flakes of bone attached to the dura or muscle. Defects also can be closed by bone grafts, and the one most suitable is the osteoperiosteal graft, which includes the periosteum and outer table of the skull taken from a normal area. It is necessary that the edges of the bone in the defect be freshened before the graft is transferred to the defect. The periosteum covering the transplant should be cut larger than the bony graft, in order that it may be sutured to the periosteum about the defect. Tibial and rib grafts can be employed, but are found to be more difficult to handle and to shape in such a way as to fill the defect.

The Treatment of Late Acute Intestinal Obstruction.

ROBERT ELMAN (*Surgery, Gynecology and Obstetrics*, February, 1933) describes the treatment of late acute intestinal obstruction. He is concerned almost entirely with the late and therefore serious cases, which offer the greatest problem in treatment. The patient seen early generally does very well, and such cases carry a low mortality, even after a radical operation for relief. It is obvious, therefore, that the education of the public as well as of the medical profession in early diagnosis would reduce the tremendously high mortality. Moreover, if the use of purgatives for abdominal pain were generally avoided, the treatment would also be easier and more favourable. Unfortunately we cannot wait until such education becomes effective. The mortality of intestinal obstruction is still around 60%, in spite of the great amount of research that has been done on the subject. All patients with intestinal obstruction receive several litres of combined solution at once. Gastric lavage is carried out. There is no objection in most cases to the use of a simple low enema to assist in ascertaining whether an obstruction is actually present. The use of morphine occasionally relaxes a patient sufficiently to allow the obstruction to subside. The author believes one should not, in general, delay operation more than a few hours in the expectation of any spontaneous release of the obstruction. The danger of increasing distension, and with it the possibility of strangulation and gangrene from delay, is too great to justify much procrastination. Operation should, therefore, be done within a few hours, unless obvious improvement is in progress. If a strangulated, and especially gangrenous, bowel is present, the origin of symptoms is easy to explain and the exteriorization of the non-viable gut is obviously indicated. To resect immediately and to make an anas-

tomosis carries a high mortality unless the duration of the disease is short, say, twelve to eighteen hours. Though the operation may be easily and quickly performed, these patients too often die shortly after the operation from what the author believes is a sudden release of distension. In the late cases, therefore, the non-viable bowel is simply exteriorized for later resection and plastic repair, and the obstructed bowel above is decompressed gradually by fractional drainage of its contents. The cause of death in untreated complete high obstruction (stomach and duodenum) is probably a physico-chemical one, due to a depletion of water and salts from the blood into the vomitus or obstructed contents. The resulting dehydration can explain all of the so-called "toxic" symptoms. Treatment with a modified Ringer's solution effectively restores the blood to normal, lessens symptoms, and permits adequate surgical treatment without great risk. The cause of death in low intestinal obstruction (ileum and colon) is probably different, but as yet there is little convincing evidence that a "toxæmia" is present.

Inflammation.

GEORGE LENTHAL CHEATLE (*Surgery, Gynecology and Obstetrics*, February, 1933) discusses the value of Burdon Sanderson's definition of inflammation. The definition is much too wide, and as it stands would include many processes of infection, local and general immunity, phagocytosis and the repair of damaged tissue, but poorly understood in 1872. In 1858, Lister had published his classical investigation upon "The Early Stages of Inflammation". John Hunter carried out many experiments to determine the nature of the "greater power of action" in inflamed tissues. The author has repeated Hunter's experiment of transplanting cocks' spurs into the combs, and in addition some were transplanted into the cervical subcutaneous tissues. In both situations greater vigour of growth was observed. The author believes that the time has come to exclude repair and the formation of neoplasms of all kinds from the subject of inflammation. The author has been surprised at the results of some series of experiments carried out, and notes with interest the organized and beautifully controlled repair of tissues. His first series comprised all the stages of a healing fracture exemplified in the ribs of guinea-pigs from the moment of injury up to thirty-six days. His next series concerned the tissue reactions following upon the ligation of the common carotid arteries of cats. The author had previously supposed that the lumen would be diminished by the organization of blood clot, whereas it occurred by proliferation of the intima. Another instance of apparently "inherited function" is the purposeful growth of cells in the formation of adhesions. The author next joins issue with the practice of refer-

ring to breast fibro-adenomata as inflammatory. Likewise the papillomata which form in the ducts of the breast have no aetiological relationship with inflammatory processes. Finally, in dealing with abscess formation following upon the injection of microorganisms into the subcutaneous tissues, the establishment of immunity transcends in importance any concurrent process of inflammation that may be present. Tissue changes which occur around an abscess could not occur if the microorganisms were able still to cause the death of tissue they manifested on their first introduction. Finally, infection and immunity, and not inflammation, are the important factors the body is concerned with in the formation and cure of an acute abscess.

Carcinoma of the Oesophagus.

WILLIAM L. WATSON (*Surgery, Gynecology and Obstetrics*, May, 1933) discusses carcinoma of the oesophagus. Much original thought, experimentation, and imagination have gone into the development of the many varied forms of treatment which have been advocated at one time or another for the cure of cancer of the oesophagus. Noteworthy among these methods was the advent of an intracæophageal method of irradiation by radium. This method was first proposed in 1904. It has fallen into disfavour. A great deal of attention has been directed towards the development of a procedure for radical removal of carcinoma located in the oesophagus. Unfortunately, surgery has not fulfilled its early promise. A careful study of the clinical course of the disease, the anatomy of the organ involved, and the *post mortem* material available will show that patients with cancer of the oesophagus should be given palliative treatment as a routine measure and that only in the very unusual early case should intracæophageal irradiation or radical surgery be attempted. The cured patient will be a medical curiosity. Intimate knowledge of the anatomy and histology of the oesophagus is essential to the understanding of the treatment problems of cancer in this organ. The exact aetiology of oesophageal carcinoma is still obscure, but definite predisposing and exciting factors are known and should be an aid in prophylaxis. Cancer of the oesophagus accounts for between 3% and 10% of all the carcinoma deaths. The disease is most frequent in the lower third of the oesophagus. Adenocarcinomata of the oesophagus are more slowly growing and give symptoms earlier than do the squamous cell lesions. Of the cases coming to autopsy 48% showed no evidence of metastases. Bronchopneumonia was a cause of death in 48% of the cases. Radical surgery of the oesophagus is indicated in a few early cases. The routine treatment should be palliative. Gastrostomy followed by external irradiation offers the most satisfactory palliation. Prophylaxis should be stressed.

Special Abstract.

DENTAL CARIES.

AN extensive review of the problem of dental caries has recently been published by Dr. Theodore Rosebury.¹ Since the subject is one to which medical practitioners have probably not paid sufficient attention, the following abstract is published in the hope that interest will be stimulated.

The discussion of dental caries has been made difficult by the practice of many authors of describing as caries many dental conditions that should not properly be so described. It must be clearly understood that caries presents a definite and specific clinical and pathological picture. It is not merely a "hole in a tooth" nor is every "hollow" tooth necessarily carious. Dental caries occurs in two main forms, the aetiology of which may be distinct. The first variety occurs in the teeth of adults and the aged, and commonly attacks smooth surfaces of the crown or exposed root, producing a large aperture at the surface and penetrating slowly. The second variety attacks children and young adults, and its greatest incidence is during the interval of transition from deciduous to permanent dentitions. The lesion first appears clinically as an enlarged developmental pit in the enamel of the crown or as a friable spot in the enamel at the contact point, in either case admitting the end of a fine-pointed instrument. Later softening and cavitation occur in the underlying dentine, spreading so as to undermine the enamel, in which the aperture may remain small for a considerable time. The process then advances along the dentinal tubules, producing a characteristic flame-like picture of penetration. The dentine is first softened, presumably by decalcification, and then undergoes digestion to form the clinical cavity. Later the pulp becomes exposed and infected, and alveolar abscess and other root infections may follow. The second variety is by far the most common and is the only one discussed by Rosebury.

Caries is a peculiarly human disease, although similar lesions have been observed in dogs and produced experimentally in rats. There is no satisfactory evidence that its incidence increases during pregnancy or in the course of certain systemic diseases, such as diabetes, and while it is probable that careful oral hygiene is an important factor in prevention, the phrase, "a clean tooth never decays", cannot yet be accepted as true.

Caries commences at points on the tooth surface that are so situated as to be out of contact with opposing teeth in occlusion. These regions accumulate food debris, partly because of their architecture and partly because they are protected from the normal cleansing movements of food and of the oral tissues. They are also the most difficult to reach with a tooth brush.

Caries occurs most commonly in the molars, next in the upper incisors and in the premolars, and only rarely in the canines and the lower incisors. Interdental caries in one tooth generally spreads to the tooth in contact. There are no inflammatory changes in caries in itself, since enamel and dentine, being non-cellular tissues, are incapable of any cellular reaction to injury. Additional dentine is, however, generally formed at the expense of the existing pulp tissue and frequently delays the exposure of the pulp.

Aetiology.

Miller's chemo-parasitic theory of caries provides the most satisfactory working hypothesis of the mechanism of its onset. Its cause is not so clear. Miller's doctrine postulates: (a) the retention of suitable carbohydrate food particles at the sites mentioned above, (b) their decomposition by bacterial fermentation, (c) the local formation of organic acids, chiefly lactic, in contact with enamel, (d) the subsequent dissolution of enamel. After the decalcification of enamel by acids its delicate frame-work of keratin is readily destroyed by the slightest friction. Dentine, however, requires both demineralization by acids

and digestion of its organic matrix by putrefying bacteria. Plaques of mucin on the teeth assist the retention of food debris and bacteria, and have been shown to be much more freely permeable to dextrose than they are to salivary buffers. Miller's explanation, however, supplies no reason for the varying susceptibility of different persons to dental caries. Some workers have regarded this as a manifestation of some systemic disorder, others as that of a purely local process.

The systemic disorder theory may be subdivided into the hypotheses that presuppose variations in the composition of the saliva and those which postulate agencies operating through the tooth itself from within. Many workers have sought, usually without results, variations in the ability of saliva to neutralize acids formed at the tooth surface. Moreover, it has been shown that the salivary buffer capacity and pH do not run parallel with the incidence of caries, and it is now probable that even the most acid saliva has sufficient buffers to make it able to neutralize local acids if it can but gain access to the region of their production. Others have advanced a view, based on clinical observation chiefly, that a deficiency of phosphorus causes some change to take place in the saliva that removes its normal inhibiting action. This view, however, has since been modified and is made less probable by the fact that the animals observed seem to have suffered from a type of dental decay distinct from caries.

Bödecker has suggested that blood buffers diffuse from the pulp outwards to neutralize acids formed at the tooth surface. Susceptible persons are supposed to be deficient in blood buffer capacity, in pulp activity, or in permeability of the dental tissues. Reliable evidence makes it unlikely that a deficiency in blood buffers can be correlated with the incidence of caries. There is little evidence that structural pulp changes may be an important predisposing cause of caries, and it has been shown that caries may occur in the rat in the presence of a histologically normal pulp. It is not known whether dental permeability to inorganic radicals varies correlatively with susceptibility to caries. That it does not is rendered probable by the discovery that dental permeability (as well as pulp activity) decreases with advancing age, as does susceptibility to caries.

Pregnancy certainly may induce structural changes in the teeth of both mother and young of experimental animals when the diet is deficient, but that these changes necessarily predispose to caries has not been verified; and it has been shown that structural dental defects need not be associated with caries. The clinical evidence is conflicting as to the increased incidence of caries in pregnant women, and there is nothing whatever to show that pregnancy in itself produces caries.

In recent years caries has been frequently attributed to vitamin deficiency diseases, and especially to rickets or, more specifically, to a deficiency of some part of the calcium-phosphorus-vitamin D complex. We know that teeth are structurally very sensitive to such a deficiency, and the view has been advanced that the defects thereby produced predispose to caries. Experimental work on this subject has, however, not distinguished between true caries and other destructive dental lesions. Clinical work with children does not indicate that rickets-producing diets are an important factor in the causation of caries. Children receiving standard diets with added vitamin D have shown slight but definite reduction of caries compared with a controlled group on the same diet but without the added vitamin, while certain workers have found little reduction of caries in a group similar to the former, but a greater reduction in one irradiated with ultra-violet light. While these findings do not seem to warrant the conclusion that vitamin D is the determining factor in susceptibility to caries, they do indicate clearly enough that its incidence may be reduced by suitable regulation of the diet. Furthermore, diets which have produced rickets in rats have failed to produce caries in these animals, although they are susceptible to that disease. Finally, an investigation of the relationship of obvious rickets in children to the incidence of dental caries failed to reveal any correlation.

¹ Archives of Pathology, February, 1933.

Vitamin C deficiency has also been thought important in relation to susceptibility to caries. It has been clearly shown that it produces profound changes in dental structure. However, in experimental work its importance in regard to caries has been assumed on the basis of its power to produce a generalized softening of the teeth in guinea-pigs. But this is not dental caries. Moreover, experiments on monkeys have been claimed to support this theory, although the apparently typical caries that resulted was produced in association with diets some of which lacked vitamin C while others contained large doses of orange juice. Hanke formerly claimed remarkable results from feeding children with orange juice, but he has now modified his views and has joined the other workers in advocating a well balanced diet for the reduction of caries in children. It may be further noted that carious human teeth do not show scorbutic changes, although these are characteristic; and even if orange juice did prevent caries, caries might still be due to the influence of some other ingredient in this complex mixture other than vitamin C. Finally, the incidence of caries has not been found to be increased in clinical scurvy.

The rôle of other vitamins in preventing caries has not been thoroughly studied. Lesions similar to those of caries in adult man have been produced in rats on vitamin A-deficient diets, but an increased incidence of caries was not found in a series of children suffering from exophthalmia. Dental lesions, unlike human caries, have been produced in rats on a diet of low pH and deficient in vitamin B. But this is not evidence that such a diet can produce caries.

Caries has recently been found to be capable of being produced in rats fed on suitable adequate diets and normal in growth and appearance. We may therefore conclude that, although dietary deficiency may doubtfully increase the incidence of caries in these animals, such deficiency is clearly not a determining factor in its production. This is in accordance with the findings of the workers engaged in child feeding experiments. The systemic approach therefore limits us to the conclusion that diet, in some manner apparently not depending on the deficiency of any ingredient, plays a definite, perhaps a determining, part in susceptibility to dental caries, and that the incidence of caries in children may be reduced by regulation of the diet.

The hypothesis that the production of caries is dependent only on local factors has on the whole been more productive of fact than the systemic approach. Attempts have been made to correlate the varying susceptibility of teeth to caries with variations in their hardness. Teeth have been classified into "sclerotic" and "malacotic" types, and the latter have been considered more liable to caries. It is, however, clinically certain that "hard" teeth may decay, and although it is probable that "soft" teeth may decay more rapidly than others, yet it is doubtful whether the structure of a tooth is of any real importance in determining whether caries will or will not attack it. Some workers emphasize the relationship of defects in the enamel to caries, while others insist that clinically hypoplastic are, in general, no more susceptible to caries than apparently normal teeth.

The possibility of the local influence of diet on caries has attracted much attention. One school has incriminated a high carbohydrate diet as directly providing pabulum for fermenting bacteria. The possibly deleterious effect of sweets on the teeth of children has been widely considered, and Bunting found that rats fed an adequate diet with added "fudge" developed caries. But there is evidence, on the other hand, that a diet consisting largely of dextrose and starch need not produce caries in animals. Work remains to be done on the possible influence of the kind of carbohydrate present in the diet.

It is well known that certain races, such as the Maoris, African tribes and Eskimos, are often free from caries when living under "natural" conditions, and develop it when conditions of civilization are introduced. This has suggested the possibility of a direct influence of the physical character of the diet on the production of caries. The view has been advanced that caries results from the soft, cooked diet of civilization, which lacks the detergent

action on the teeth of the coarse, more fibrous "natural" diets. Experiments conducted on men to test this theory were, however, disappointing, as it was found that a so-called "sticky" diet left the teeth cleaner and produced no more abundant bacterial growth in the oral debris than supposedly detergent diets. The influence of the size of cereal particles in adequate diets on the production of caries in rats has also been investigated. Diets containing coarser particles have produced it, while those with finely ground particles have failed to do so. Cereal particles of different size, of course, also differ in phosphorus content, but it is clear that this is not a factor in the production of this lesion. Whether this discovery of the importance of cereal particle size can be applied to human caries is as yet undetermined.

As was mentioned earlier, there has been a tendency among dental investigators to apply the term caries to any destructive dental lesions in rats except those obviously due to trauma. In this paper the term, particularly when applied to findings in animals, is reserved only to such lesions as closely resemble the dental caries previously described as a clinical and pathological entity. Therefore, if studies in rats are to have any application in human caries, the following criteria for the recognition of true caries must be established. The lesion must: (i) present a histological picture similar to that in human teeth, (ii) begin in the enamel, (iii) occur in young animals, and (iv) progress with relative rapidity. Many of the lesions in rats hitherto reported as caries began in dentine, generally at the summit of the molar cusp, where dentine is normally exposed in the rat, occurred in adult animals only, and seemed to follow a chronic course.

Bacteriological Investigations.

Some of the most striking results in experimental research in dental caries have been obtained from a study of its bacteriology. Miller's doctrine was originally based on his discovery of many bacteria in decayed teeth. This was later confirmed and the view developed that the process might be initiated by any of the group of fermenting bacteria. In 1903 a Gram-positive, non-sporing rod was isolated, which was probably identical with the lactobacilli mentioned below. In 1915 certain workers emphasized the occurrence of *Bacillus acidophilus* and thread forms, and the great increase in numbers of these organisms with the development of caries. In 1917 these findings were confirmed, and it was reported that the so-called Moro-Tissier group of bacteria—*Bacillus acidophilus*, *Bacillus bifidus* and others—preponderated in caries. Later, it was shown that acidophilus-like organisms are the only forms that develop consistently in cultures from carious teeth in a carbohydrate broth of pH 5 and it was thought that the acidogenic powers of these organisms were sufficient to decalcify enamel. Work in America in 1925 showed that: (i) lactobacilli are uniformly present in carious teeth, (ii) lactobacilli are frequently absent from non-carious mouths, and (iii) the presence of lactobacilli in a non-carious mouth may foreshadow the appearance of clinical caries on later examination. In the same period certain English workers isolated a non-hæmolytic streptococcus (*Streptococcus mutans*) from carious teeth, and considered it more important than *Lactobacillus acidophilus* as a cause of caries. American workers, however, consider *Streptococcus mutans* to be a variant of *Lactobacillus acidophilus*, which shows coccic forms frequently, since true streptococci are rarely found by them in cultures in mediums of pH 5. In spite of these considerations, there are serious objections to the view that dental caries is a specific infectious disease with *Lactobacillus acidophilus* as its ætiological agent. The organism may often be obtained from mouths without caries, although reduced in numbers or in acid resistance or in both. It has been fed to caries-free persons in the form of massive doses of milk cultures without producing caries, and it has also been fed to animals without effect. Rats constantly have organisms of this group in their mouths without developing caries, and when caries is produced in these animals, the lactobacillus flora is not appreciably altered. Finally, the feeding of lactobacilli from human

caries to rats on caries-producing diets does not increase the incidence of caries in their teeth. Therefore, at present we must conclude that factors other than infection are of determining importance in susceptibility to caries.

Although lactobacilli are much more strongly acidogenic than other strong fermenters (*Staphylococcus aureus* and *Bacterium coli*), yet some workers still hold that many bacteria are potentially capable of decalcifying enamel. It was once believed that enamel does not dissolve at pH levels much above four, which would be well in accordance with the concept of specificity of lactobacilli, since they alone can reduce the pH of artificial mediums below this level. Later work has, however, shown that the solubility of enamel depends not only on the pH, but also on the salt (particularly calcium and phosphate) concentration of the solvent, and that it may dissolve slightly in neutral or even alkaline solutions. Saliva contains sufficient calcium and phosphate, even when the pH is low, to prevent solution of enamel, unless it is prevented from reaching the tooth surface.

The possibility of an immunological factor in the problem of susceptibility has also been considered. Slight and inconsistent evidence has been found of the presence of normal agglutinins to lactobacilli in the salivary caries-free persons. However, the occurrence of lactobacilli in the mouths of men and of rats without caries makes it improbable that the presence of an inhibiting principle in saliva can determine non-susceptibility. A recent report states that agglutinins to lactobacilli are generally present in the serum in greater concentration in caries-free persons than in those with caries, and that susceptible persons generally show a cutaneous reaction on intradermal injection of filtrates of lactobacillus cultures to a greater extent than those who are caries-free. The significance of these findings, however, awaits further study. The procedure now universally employed in the treatment of caries is based on Black's doctrine of extension in preparation of the cavity. It has been invariably found that caries does not recur locally when a cavity, after the removal of damaged tissue, is extended beyond the limits of the "out-of-contact" areas to regions that are "self-cleansing", then suitably disinfected and filled with an inert durable material placed so as effectively to seal the margins of the cavity. A method of preventing dental caries, "prophylactic odontotomy", involves the operative removal of the susceptible areas in the mouths of children and their replacement with fillings as soon as practicable after the eruption of the teeth. Although this method has been widely and rather successfully employed, its universal application obviously presents serious practical difficulties.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on May 25, 1933, Dr. A. HOLMES A COURT, the President, in the chair.

Pneumonokoniosis.

Dr. J. G. EDWARDS, on behalf of Dr. W. A. EDWARDS, read a paper entitled: "Pneumonokoniosis" (see page 131).

Dr. C. G. McDONALD, who opened the discussion from the clinical aspect, began by congratulating Dr. W. A. Edwards on the soundness of the views put forward. Dr. Edwards had delivered an excellent review of the present state of our knowledge of pneumonokoniosis, and had shown an admirable restraint and conservatism in his treatment of the subject. In Australia two occupations only, namely, rock-chopping and coal-mining, were of great medico-legal interest from the point of view of pulmonary fibrosis. The first occupation, rock-chopping, and its allied trades, led sooner or later in the majority of workers to silicosis. There was little difference of opinion as to the

gross incapacity which the silicosis of rock-choppers, sewer workers and metalliferous miners could produce, and all were agreed more or less on the mechanism of its production. Very few would object to the statement that silicosis was due to silica. Dr. McDonald wished to emphasize only two clinical points. The first was that silicosis sometimes developed extremely slowly, and might produce harmful effects very many years after the worker had ceased to be employed in his dangerous occupation. He illustrated this by reference to a patient who had attended the Anti-Tuberculosis Dispensary at the Royal Prince Alfred Hospital in August, 1931. This man had worked in gold mines in Western Australia from 1895 till 1907, a period of twelve years. In 1907 he had accepted employment as a linesman in the New South Wales Department of Tramways. He was able to do this work without any inconvenience, and had enjoyed apparently excellent health till June, 1931, when he first noticed dyspnoea on exertion. The dyspnoea had gradually become worse, and he had since been troubled with a distressing cough. Dr. H. R. Sear had made a radiographic examination of his chest in August, 1931, reporting the existence of very advanced pneumonokoniosis with considerable fibrosis extending upwards and outwards from the right hilum with definite cavity formation, which he regarded as probably tuberculous. That the silicosis from which this man suffered was complicated by added tuberculosis was proved by the discovery of tubercle bacilli in the sputum. The great interest of this case lay in the fact that a long latent period of twenty-four years existed between the cessation of exposure to silica and the development of the first material symptom.

The second clinical point which Dr. McDonald desired to emphasize was his belief that silicosis seldom caused incapacity until tuberculosis developed. In the absence of extreme emphysema and in the absence of clinical conditions independent of silicosis, he believed that the majority of silicotic persons worked on until a complicating tuberculosis laid them low. He did not believe that cardiac failure occurred as frequently as some observers would have them believe. The extreme breathlessness presented by these patients was, in the absence of advanced emphysema, due to pulmonary and not to cardiac complications.

Dr. McDonald then went on to a consideration of coal-mining as the second great industrial hazard which led to pulmonary fibrosis. All were agreed that the pneumonokoniosis of the coal-miner was a less serious type of fibrosis than the silicosis of the rock-chopper. All, however, were not agreed, at least in Australia, that anthracosis was in reality a silicosis. Nevertheless the majority of authorities in every country of the world had stated with one voice that anthracosis was in essence silicosis. He had been struck a few years ago by a list of opinions collated from English, French, German and other medical literature by Dr. T. L. O'Reilly and Dr. M. R. Finlayson, of the New South Wales Department of Transport, in which authority after authority emphasized *ad nauseam* the harmlessness of coal *per se* and the importance of silica only as the one hazardous dust. If all these authorities were to be believed, coal could be described at once as innocuous, and there should be an end to the contentious discussions in Australia as to its effects on pulmonary tissue. However, in Australia Dr. Badham had, during recent years, maintained that this world-wide view was wrong, and he had suggested that coal might and did cause pulmonary disablement either by virtue of its hydrocarbons or by virtue of the silicates which it contained. On the question of the hydrocarbons as a cause of pulmonary fibrosis, no strong evidence had yet been put forward to show that these substances did actual damage to the lung. There was no doubt that coal was easily eliminated from the lungs of healthy coal-miners. Further, there was no doubt that coal-miners worked on to a healthy old age after spending long periods of forty to fifty years in continuous coal-mining work without any evidence of a disabling pneumonokoniosis. Statistics, which had been quoted on many occasions, proved conclusively the long life enjoyed by miners in "pure" coal compared with the tragically short careers of the sewer workers and the rock-choppers. Whether or not coal,

by virtue of the carbon it contained, produced a fibrosis could only be an academic problem. It remained certain that the man who worked in coal alone need have no fears that his life would be terminated as a result of his service in the coal mines. Dr. Badham had taken more kindly to silicates than to carbon as a cause of fibrosis. He had maintained very vigorously that the common view that combined silica was harmless was quite wrong. Much work had been done in South Africa and England on the toxicity of silica. The old view that silica particles acted mechanically on the lung, stimulating fibrosis by means of their sharp edges or spicules, had long since been discarded. It was now believed, and the evidence appeared to be overwhelming, that silica produced a slow fibrosis only when it became soluble in the alkaline fluid of the pulmonary tissues, forming a silica sol or an actual silicate. Only particles less than ten microns in size could be seized by the alveolar phagocytic cells and carried into the lymphatics to be acted on by the alkaline fluids. The discovery of the chemical action on silicon oxide had represented a brilliant advance in industrial pathology. It was now believed that this very chemical action on silica stimulated the production of fibrous tissue in the lung substance. Preformed silica compounds, such as silicates, did not produce this specific action. If, therefore, Dr. Badham's contention was right, the whole fabric of the modern pathology of silicosis tottered and fell. The only doubt in regard to the harmlessness of silicates was prompted by the condition of fibrosis found in asbestos workers, but the problem of asbestosis was not merely a problem associated with the direct action of silicate. It was quite possible, as the speaker's namesake had suggested, that asbestosis was simply a special form of silicosis, the magnesium silicate present in the asbestos being decomposed chemically within the lung into magnesium and free silica, which latter became acted on by the pulmonary tissue fluids in the same way as in cases of frank silicosis.

Dr. McDonald then made mention of Professor Lyle Cummins, a very sound observer, who had advanced a few years ago the hypothesis that coal dust could cause incapacity when the pulmonary lymphatics had been already injured by preexisting inflammation of the pulmonary structure or by preexisting fibrosis due to the more noxious dusts, notably silica. Previously, Cummins had elaborated this view in his earlier published papers, and it had been seized upon by those who seemed anxious to prove that coal-mining was a dangerous occupation. But in his more recent publications Professor Cummins appeared, like a good soldier, to have retreated gingerly, but not completely, from his former view. So far as the speaker could judge, Cummins's present opinion was that coal-miners might be incapacitated by the inhalation of coal dust only if and when they had suffered from a preexisting silicosis. There was therefore little justification for the assertion frequently made now in Australia, on the alleged authority of Professor Cummins, that a man who had worked on coal only all his life might be seriously incapacitated by that hazard. Professor Cummins had also originated the hypothesis of so-called non-tuberculous cavitation of the lungs, a pathological condition of which they had read much recently from the pen of Dr. Badham. That small ragged breaks in the lung structure occurred in areas where coal had accumulated in great amount was true enough, though many of them appeared to be *post mortem* lacerations. Where large definite cavities were found in the lungs, whether at the apices, in the hilar regions or at the bases, no other presumption could, in the light of our present knowledge, be made than that these were tuberculous. The fact that tubercle bacilli were often not found in the sputum or in the pulmonary structure *post mortem* proved nothing. Tubercle bacilli were notoriously hard to find, and the existence of a few giant cells on microscopic examination of the tissue, together with fibrous tissue formation and *endarteritis obliterans*, was strong presumptive evidence of the tuberculous nature of the lesion. After all, there were very few pathological conditions which could produce cavities. Far and away the most common was tuberculosis, and the others were pulmonary abscess and bronchiectasis.

Dr. McDonald's next statement was that if it was maintained that both the silicates and the carbon compounds of coal were innocuous, it had nevertheless to be admitted that coal-miners did suffer from pneumonokoniosis—a very slow and a comparatively mild fibrosis as a rule, but in rare instances definitely and completely disabling. What, then, was the cause of this incapacity? New South Wales coal contained on an average only 0.5% of uncombined silica. This silica content was not sufficient to cause disablement, and therefore lifelong miners in "pure coal" could not, he believed, develop any incapacitating fibrosis. Physicians were apt, however, to forget that miners frequently cut through seams of coal in which bands of stone were present, and that those miners who machined coal frequently tore up considerable quantities of stone dust from the mine floor. It took a coal-miner thirty to fifty or more years to show any serious degree of fibrosis, and in that period much silica was inhaled and slowly converted into silica sol in the lung structure. Anthracosis therefore was essentially silicosis, but one whose development was extremely slow. Although coal tended to inhibit the development of tuberculosis, silica tended, on the other hand, to favour its spread. It was not uncommon to find in old coal-miners silicosis complicated by pulmonary tuberculosis, and in those instances it could be assumed that the baneful influence of silica had counteracted the immunizing influence of coal dust.

Unfortunately physicians were not always given frank statements of the working conditions in New South Wales coal mines. There appeared to Dr. McDonald to be a "silent conspiracy" between the employer on the one hand and the employee on the other. The employer was anxious to keep secret the fact that silica was a definite hazard in coal mines, lest compensation laws should be altered to bring all incapacity due to coal-mining under the *Silicosis Act*, and the employee was equally anxious to hide the same fact. This was due to our unsatisfactory legislation for the compensation of pulmonary fibrosis caused by the hazards of certain industries. It was ridiculous that a man suffering from silicosis in the County of Cumberland in New South Wales could obtain compensation, while another workman, who contracted his disease just outside the County of Cumberland, could not obtain it unless he could prove that some other dust than silica was responsible for his incapacity. As a result, medical experts were frequently tempted to disregard the hazard of silica in coal mines and to confine their attention to the alleged baleful influence of other dusts. It was a pity that any of these cases had to be fought in court. All incapacity definitely due to working conditions should be compensable, and a committee of medical men, working only in a scientific spirit, should decide whether the incapacity of any particular miner was sufficient to be compensable.

Lastly, Dr. McDonald appealed to radiologists, who were in an extremely influential position so far as the courts were concerned, to use the greatest possible discretion in the reports which they gave. The incapacity of pneumonokoniosis was a clinical and not a radiological problem, and while he had no sympathy with those physicians who held that radiologists were photographers or mechanics, he believed that radiologists often drew inferences of incapacity from radiographic appearances which no clinical examination could support. The shadows of X ray plates were extremely difficult to interpret, and he suspected that the markings described sometimes by radiologists as representing alleged late primary or early secondary stages of pneumonokoniosis were frequently not due to pneumonokoniosis at all, but to the many conditions which Dr. Edwards had described.

DR. C. BADHAM regretted that the paper of Dr. Edwards on the subject of pneumonokoniosis was not such as the well informed audience there that night might reasonably have expected. Dr. Badham found the address wanting in recent knowledge and containing statements relating to the *Silicosis Act* and activities of the Silicosis Committee which were hard to reconcile with facts. There was so much to criticize in it that it seemed useless to attempt to do so in the limited time at Dr. Badham's disposal. It would surely have been better if Dr. Edwards had con-

fined himself to that particular aspect of this subject in which he was so skilled and well informed.

Dr. Edwards said how serious it was to call a man a silicotic, and urged his fellow radiographers to refrain from doing so until a very definite fibrosis was present. Dr. Badham suggested that it was equally serious to use the term tuberculosis, and he urged that, as tuberculosis as seen by the radiographer was such a varied condition, it would be to everyone's advantage if some such term as infective consolidations were used.

The plea he wished to make was for the need for greater efforts in pathology. Unfortunately radiology had put a blight on the study of dust diseases. The radiographic report at present swamped the field, shackled the physician and left the pathologist lamenting lost opportunities; if the present position continued, it would be to the disadvantage of all parties. So the plea which he wished to make was one for far greater efforts to be made in the study of pathology of dust diseases. He asked them all to try to help him to secure lungs for examination, and he mentioned in support of his claim that his family had a vested interest in diseases of the lung. In 1814, Dr. Badham's great grandfather and namesake had described and named bronchitis, and in asking support for the old firm, even if it had fallen on somewhat evil days, Dr. Badham assured them that only one interested in this matter would be able to gather together sufficient material and would find time for its study.

Dr. Badham then referred to the cost of compensation for dust diseases. He said that it cost the country more to pay compensation for dust diseases at Broken Hill than to run the Royal Prince Alfred Hospital. Over £1,000,000 had been paid out in twelve years at Broken Hill, and the pathological work done was very little, and this was deplored by Dr. George, who was in charge of the Medical Bureau. In the regulations of the silicosis scheme Dr. Badham had placed a clause which gave power to the medical authority to conduct *post mortem* examinations on all beneficiaries, but this power was not being used, and though twenty-six persons had died, only four or five *post mortem* examinations had been made. This scheme had already cost the country over £60,000, and the pathological work was practically nil. The scheme had not contributed to the knowledge of silicosis.

Now it had been written recently that Dr. Badham was not a radiologist. Shortly it would be said that he was not a pathologist, and in a little while that he was not a clinician; but he said that a competent industrial hygienist was all three, and, specializing as he did in the limited fields of industrial poisoning, dust diseases and ventilation, he could look for and had in other parts of the world attained success. If Dr. Badham's chest radiology was poor, then all he could say was that the radiologists who had tutored him for ten years must have found him a dull fellow, and he thanked Dr. Edwards and others for their unrequited labour.

He wished to show three cases, not to discomfort anyone, but in order to stress the need for pathological work. In two of these, Cases V and XXII (the recent coal-miner's lung paper by Dr. Taylor and himself), an eminent radiologist had diagnosed silicosis and tuberculosis, yet no evidence of tuberculosis could be found *post mortem*. The third one was that of a sewer miner, said by the radiologist to be a case of tuberculosis only, yet found *post mortem* to be a case of massive silicotic fibrosis. Neither portions of a small cavity in a consolidation nor of glands of both lungs injected into guinea-pigs produced tuberculosis. The chemical analysis of the lung revealed the presence of an amount of free and combined silica such as was found in their cases of gross silicosis.

Now, if these cases were found in their limited series of lungs, was it not very reasonable to urge pathological examination of the lungs of cases of dust fibrosis?

On the chemical analysis of the lungs Dr. Badham laid great stress, and a study of the table in his and Dr. Taylor's last work must prove increasingly interesting to all of them. He often said that the best dust sampler was a pair of human lungs.

In conclusion, Dr. Badham referred to South Africa and animal experiments. He said that in South Africa he

had found that a much broader outlook was taken than with them in Australia, where the report of the radiographer had been allowed to dominate such factors as clinical signs and histories and industrial history. In South Africa in particular a man's mining history was given far greater weight. Many cases which in Australia would be called simple tuberculosis were placed in the silicotic class. Dr. Irvine's statement was that they attempted only a rough justice, but Dr. Badham thought this rough justice a better thing than the alleged ability of our radiographers to diagnose simple tuberculosis in a worker exposed to silica dust. Moreover, the pathological evidence which they showed supported their views, and Dr. Badham had seen lungs with consolidations proved by biological examination to be non-tuberculous; yet the skiagrams of these lungs would be interpreted in Sydney as demonstrating silicosis and tuberculosis. The Watkins-Pitchford idea of a tuberculous basis for silicosis had been abandoned, and Gardner was able to show in the lungs of animals that silicosis was a separate entity, and Simpson and Strachan had demonstrated the same fact in regard to human lungs.

In regard to animal experiments, Haynes had recently found the following dusts to produce harmful reactions in the lungs: flint and shale, kaolin, slate, precipitated silica, wood charcoal, chalk, aluminium hydroxide, calspar, emery, magnesium carbonate, colloidal coal and shale in massive doses. His work suggested that the disilicates and trisilicates were responsible for tissue reactions.

Kettle had devised a method to determine the pernicious character of any dust by injection into subcutaneous tissue. A coagulation necrosis of tissue resulted, followed by specific changes with dusts which were active. He had shown that silica, shale, kaolin (a disilicate), asbestos were active, but that silica coated with iron was inactive. This inactivation of silica by coating with iron might be in line with a fact which they had observed in their coal miners' lungs—the relative insolubility of silica coated with coal. Gardner this year hoped to publish a newly devised method for the rapid detection of biological activity of inorganic dusts; he also forecasted some very interesting work on the mode of action of dusts such as granite. A new era was dawning in the study of dust diseases.

Dr. L. L. HOLLAND thanked Dr. Edwards for his paper and Dr. Badham for his demonstration. He pointed out that medical practitioners in Australia were liable to fall into the danger of forming false ideas from false premises. He had had a large mining experience and had observed that many of the men claiming compensation had not always worked in Australian mines. In England men started working at a much earlier age, and it was possible for a man to arrive in Australia at the age of twenty-one having had eleven years' experience and having worked under conditions different from those in this country. Therefore their ideas would be considerably modified when they were dealing with people who developed pulmonary conditions in this country, but who had not always lived here. It was possible that when they arrived the damage had already been done. Haldane had said that chronic bronchitis was more common in England among coal-miners than among others. Dr. Holland had seen many coal-miners and would say that in Australia the incidence of bronchitis was no higher, or that the condition was even less common among coal-miners than among ordinary people.

Dr. Badham's method of attack in dealing with Dr. Edwards's paper was typical of the way in which they were wandering in the darkness. The hiatuses in their knowledge, mentioned by Dr. McDonald, were only too great. Dr. Holland made a plea for greater cooperation between all concerned, the coal-miner, the mine-owner, the pathologist, clinician and radiologist; only in this way could progress be made. The pathological aspect was eminently important; but the work must be done by experts. Just as it was possible for coal dust to modify a tuberculous lesion, so it was possible for a tuberculous infection to modify the condition of a lung affected by coal dust. It was time that they got on with the work. Cooperation must replace the recriminative attitude evident that evening.

DR. MAY HARRIS thanked Dr. Edwards for his able paper, which she had found most helpful. She had had some experience of this disease among coal-miners. In the first place the condition was one of fibrosis, by which the men were not disabled. But if later on tuberculosis set in, the men did become disabled. It was the tuberculosis that was the disabling factor in all these cases. Dr. Harris could not agree with many of Dr. Badham's deductions. Anthracotic cavities resembled old tuberculosis cavities. The fact that no tubercle bacilli were demonstrated in the sputum might be owing to the bacillus existing in a state in which it was not acid-fast and in which it was stained by methylene blue. The chromophobic state had been proved; it could be brought back to the acid-fast state.

DR. H. R. SEAR opposed some of the views expressed by Dr. Edwards. He stressed the importance of fluoroscopy and quoted the case of a man who had been stated to be suffering from a tuberculous lesion. He could find no evidence of a tuberculous lesion, but fluoroscopy of this man showed an extraordinary condition to be present, the curve of the pulmonary artery being exaggerated and the hilar shadows greatly enlarged and pulsating, as also were the main vessels of the lungs. Such a condition could not be detected in the skiagram alone.

Again, the statement that the appearance of the nodular stage was essential in the making of a radiographic diagnosis of pneumonokoniosis was inaccurate. He referred to the work on asbestosis in which the causative dust was a silicate. In this condition death occurred rapidly, with the classical symptoms and signs of pneumonokoniosis; and in it, as had been pointed out abroad, cases often went on to death without the appearance of the nodular stage, but only with a fine diffuse fibrosis. In these cases fluoroscopic examination was essential and it had been maintained abroad that an opinion based on evidence provided by skiagrams alone would frequently be fallacious.

DR. COTTER HARVEY remarked that what had been said only made them realize how difficult the subject under discussion was. As a clinician he was particularly interested in the subject, but he believed that it was incapable of solution in this country for a number of years, because the radiologists differed in their interpretations of the pictures taken. Irvine, in a recent paper, stated that a technically good radiograph taken instantaneously was the most reliable single criterion in the diagnosis of silicosis. Later, in the same paper, he admitted that skiagrams could also be most misleading. Silicosis was a progressive disease, even when the patient was away for years from dusty work, and it might be the result of work in silica years before it became manifest. Dr. Edwards had said that ten years was the minimum time for getting silicosis. Dr. Harvey would rather say that it was the average period after which a person exposed to silica for some years developed silicosis. Fibrosis was not related to incapacity. Radiology had not yet reached a state of precision that would enable the radiologist to state the degree of incapacity of the patient. The only certain feature indicating tuberculosis was tubercle bacilli in the sputum.

Dr. Harvey mentioned a case and illustrated it with skiagrams, of a patient who had been diagnosed in 1925 as suffering from advanced pneumonokoniosis with no tuberculosis. The patient was a rock-chopper of fifty-nine years and was still working; he was prepared to keep on working. Three years later his condition was still reported to be pure pneumonokoniosis and he was given compensation of three pounds per week. Later on he had hemoptyses and a spontaneous pneumothorax occurred. In 1930 there was evidence of calcification, and in 1932 the radiologist said that a healed tuberculous lesion was present with calcification. There was now no radiological evidence of silicosis. The points of interest were that the patient had been able to work in spite of the fibrosis present; that the radiological appearances changed so completely; that there were no tubercle bacilli in the sputum, and that during six years the patient gained weight and felt well.

Dr. Harvey also showed several other skiagrams. Nodular fibrosis was found to occur in a man who had never been near stone dust, yet his lungs presented a good picture of pneumonokoniosis.

Another patient had been told by the Silicosis Commission that he had tuberculosis and was therefore not eligible for compensation. He went to the Repatriation Department, where he was told that he had only silicosis. After he was shuffled for two years between the two departments, the Repatriation Department yielded and agreed to give him a pension. He had fine, discrete, nodular fibrosis.

Finally, Dr. Harvey showed skiagrams of the chest of a patient who might have been suspected of silicosis; on the other hand, the condition might have been military tuberculosis. The patient was known to be a woman of twenty-five years who showed extensive nodular infiltration. Serial radiographs showed a progressive diminution in the infiltration, until eighteen months later the condition had entirely cleared up. The patient kept well for two years, but the condition had recurred, and a skiagram was shown identical with the first one taken. This occurred fifteen months ago, and the process was once again resolving. Dr. Harvey had no idea what was the diagnosis. He showed the various films to indicate how difficult was radiological diagnosis in dust diseases.

DR. K. E. SHELLSHEAR expressed his pleasure at having heard Dr. Edwards's paper and the discussion that it had brought forth. The diagnosis of pneumonokoniosis in coal-miners was not a difficult one if nodules were formed. But it was impossible if nodules were not formed. From the point of view of the diagnosis of pneumonokoniosis, once nodules were present, using reasonable care, the radiologist should know whether the condition was military tuberculosis or anything else; he should be able to diagnose pneumonokoniosis. Areas of consolidation needed investigation. In ordinary tuberculous cases they acknowledged that the areas of consolidation and cavities were nearly always towards the apex and not the base. It was remarkable that areas of consolidation in pneumonokoniosis were always in the same position.

There was no proof that silicosis, anthracosis or coal dust could cause consolidation or cavity formation, apart from tuberculosis; it was more or less an assumption. This work needed much investigation. So far, most evidence was in favour of the cavities being tuberculous.

DR. M. R. FINLAYSON said that the last speaker's remarks had brought him to his feet to refer to some of his experience of pneumonokoniosis at Broken Hill. Dr. Badham had stressed that the lungs exhibited by him were the essential coal-miner's lung, but Dr. Finlayson considered them to be in all respects similar to some seen in metalliferous miners. He did not have the opportunity of doing a great many *post mortem* examinations at Broken Hill, as they were difficult to obtain. He found it hard to get away from the accepted idea that all cavitation was due to tuberculosis, but it stuck in his mind that there was something more to it. He had seen men who had been many years in the mine and who had marked fibrosis and large areas of consolidation; but these same men lived till they were over seventy and did not die of tuberculosis; they showed no evidence of tuberculosis and no bacilli in the sputum. Most died from cardiac failure, and even when they became debilitated there was no evidence of tuberculous infection.

It had to be admitted that their ignorance of this subject was as great as their knowledge and that a lot more pathological work had to be done. From the point of view of industrial litigation it would be very satisfactory if the radiologist would make in doubtful cases his diagnosis only after discussion with the physician.

DR. G. C. WILLCOCKS said that he was interested to hear discussed the difficulties of diagnosis. He wished to refer to the question of incapacity, which, from the patient's point of view, was the most important point. He had examined some fifty coal-miners recently, probably the worst cases in the northern and southern coalfields. Of these about half a dozen were definitely incapacitated, some owing to pulmonary disorders of uncertain causation, others owing to old age and arterial disease or heart disease. When disease of more than one system existed it was difficult to say which was the incapacitating disease.

Dr. Willcocks asked what really constituted disablement. He quoted an instance of a well nourished patient who

showed no evidence of disability, except his own statement that he was short of breath; one could not say that such a man was disabled merely because there were some markings in the X ray films of his lungs. The examiner could only get the man to exert himself and note the degree of breathlessness and the pulse response. But even this might be fallacious, for if the man knew that his claim for compensation depended on his being breathless, it was probable that both his breathing rate and pulse rate would be increased owing to nervous influences. Nothing could be more deceptive than a quick pulse due to nervousness.

The physician must consider all these points in arriving at a decision as to whether the man was or was not disabled. Too much reliance should not be placed on the X ray pictures.

Dr. H. M. CUTLER expressed his appreciation of the opinions given during the discussion, all of which represented work done. The question of incapacity was understood by him from the point of view of comparing people in the Silicosis Commission who came up for examination with those before the Pneumonokoniosis Board. Many of the former had radiological disability that was outwardly not manifest. These men could carry on; they were different from those seen in the Pneumonokoniosis Commission, who had only diffuse fibrosis, which, nevertheless, made it impossible for young men in their thirties to do anything but sit in the back yard in the sun. The radiologist could estimate the amount of damage apparent in the lung, but could not estimate the disability, though the radiological examination of the chest remained the most important single clinical factor in the examination of the chest.

Dr. J. G. Edwards, in reply, said that the paper had been prepared and read for the interest of members and that he did not expect it to be up to Dr. Badham's level. As to the legal points in this paper that had been criticized by Dr. Badham, he had as his authority an eminent member of the legal profession, to whom it would give him pleasure to recount Dr. Badham's opinion. Dr. Edwards expressed the opinion that perhaps Dr. Badham's guinea-pigs were immune to tuberculosis.

As to the time limit for the development of pneumokoniosis, Dr. Edwards said that eight years was the limit for the normal healthy individual. Before that there was a peculiar type of recurrent bronchitis that would clear up after some months.

In regard to tuberculosis at Broken Hill, he referred to the report of Dr. George after eight years. Of the first stage cases after eight years, 45% of the patients developed tuberculosis; of those in the second stage, 57% developed tuberculosis. Of the fourth stage cases two-thirds of the patients were dead in eight years.

Dr. A. HOLMES A COURT complimented the speakers on the illuminating discussion that had been contributed by members expert in their several spheres of knowledge. He had pleasure in expressing his appreciation on behalf of the Branch.

MEDICO-POLITICAL.

At the request of the Secretary of the Victorian Branch of the British Medical Association, the following resolutions of the Council of the Branch are published for the information of members.

Notification of Infectious Diseases.

Section 123 of the *Health Act* directs that every medical practitioner who becomes aware that any person is suffering from a notifiable infectious disease shall immediately: (a) notify on the prescribed form to the council for the municipal district; and (b) inform the head of the household or the occupier of the premises of the infectious nature of the disease.

Difference of opinion has arisen as to whether under that section the word "premises" refers to the premises where the patient is living at the

time of the illness or the premises where he is working. Legal opinion has been obtained to the effect that: (a) the only safe course for the medical practitioner is to notify the occupier of the premises where the patient lives, and (b) reference to the history of the section indicates that the intention was that the living premises of the patient are the premises intended to be referred to.

The health authorities of the State are of the opinion that the spirit of the section will be complied with if the practitioner notifies the council of the municipal district, giving all particulars, and, if the patient is a minor, also informs the head of the household where the patient lives.

The health authorities consider that it is the responsibility of the local authorities to take any necessary action relating to premises where the patient works or where the patient, if not a minor, is lodging.

Public Lectures.

Permission to give public lectures, including broadcasting, under schedule (g) of "Ethical Principles" shall not be given to members of the Branch unless application for such permission has been submitted to the Ethics Committee and confirmed by the Executive.

This resolution will come into force on September 1 next.

Opticians.

The Ophthalmological Section desires that members should be warned that the practice of sending patients direct to opticians for glasses is dangerous, as early symptoms of organic diseases are in that way frequently overlooked.

As this course is frequently advised with the idea of saving expense to patients, the Section desires to spread the information that both the fees paid to the specialist and the charge for glasses can be adjusted to the patient's means if the doctor sending the patient advises the oculist of the necessity for such adjustment.

Farmers' Accounts.

Members who have accounts for medical attendances against farmers who are working under Protection Certificates, are advised by the Council to send their accounts without delay to George Brown, Esquire, Secretary, Farmers' Relief Board, State Public Offices, Melbourne.

The Council has reason to believe that some action may be possible on the part of the Farmers' Relief Board to secure some payment on these accounts.

Private Contracts.

Members are advised that before entering into any definite arrangements altering conditions of medical practice, such as the initiation of contributory schemes, the appointment of honorary medical officers to public hospitals, the establishment of community hospitals *et cetera*, such suggested arrangements should first be submitted to the Council of the Branch for consideration and approval.

Lodge Certificates.

The Council has accepted the offer of the Friendly Societies' Association to pay a uniform rate of five shillings for an additional sickness certificate for a friendly society members who is an employee of the railways or other departments of the State, when the conditions of his employment require such a certificate. This agreement will apply only to a member on the list of the doctor who supplies the certificate, and must be paid for at the time the certificate is given.

NOTICE.

DR. J. G. HUNTER, the Medical Secretary of the New South Wales Branch of the British Medical Association, announces that the following books have been added to the library of the New South Wales Branch: "The Legal and Ethical Aspects of Medical Quackery", L. Le M. Minty; "Chronic Rheumatism and the Pre-Rheumatic State", J. D. Hindley-Smith; "Diseases of Old Age", F. M. Lipscomb; "Food, Health, Vitamins" (Fifth Edition), R. H. A. and V. G. Plimmer; "The Elements of Medical Treatment" (Second Edition), R. Hutchison; "Common Skin Diseases", A. C. Roxburgh; "Talents and Temperaments", A. Macrae; "Tuberculosis of the Lungs", S. L. Pipiani; "Medicine: Essentials for Practitioners and Students", G. E. Beaumont; "Gray's Anatomy: Descriptive and Applied" (Twenty-fifth Edition), edited by T. B. Johnston; "Principles and Practice of Psychiatry", A. Cannon and E. D. T. Hayes.

Public Health.

INTERNATIONAL VITAMIN STANDARDS.

In June, 1931, a conference was held in London under the auspices of the Permanent Commission on Biological Standardization of the Health Organization of the League of Nations, with the object of considering the possibility of adopting standards and defining units for certain of the vitamins. The Conference recommended the adoption of a standard and defined a unit in terms of such a standard in the case of four vitamins—A, B₁, C and D. The National Institute of Medical Research, Hampstead, London, was nominated to act on behalf of the Health Organization of the League of Nations as a central laboratory for the storage of vitamins A, B₁ and D. In the case of vitamin C no preparation of storage of a stable standard was involved by the recommendation of the Conference, which was satisfied for this vitamin to recommend the use of fresh lemon juice as a standard, and to define the unit as the anti-scorbutic activity of 0.1 cubic centimetre of the juice prepared according to a simple method described in the report.

Vitamin A.

Carotene was recommended as the international standard for vitamin A, and the unit was defined as the vitamin A activity of 1 Y (0.001 milligramme) of the standard preparation of carotene. Eight laboratories have contributed varying quantities of carotene, and these have been mixed at the National Institute for Medical Research; the mixed carotene has been purified by recrystallization until the melting point was above 179° C. The highly purified preparation has been distributed in ten milligramme quantities into small ampoules in an atmosphere of pure nitrogen, reduced to dryness, and the ampoules sealed.

Vitamin D.

With regard to the international standard for vitamin D, the Conference recommended that the standard solution of irradiated ergosterol, which was issued from the National Institute for Medical Research, Hampstead, during the years 1930 and 1931, should be adopted as the international standard. On account of the fact that this standard preparation had been intended, primarily, for the needs of Great Britain alone, it was considered desirable to prepare a larger quantity to meet the needs of other interested countries—nineteen in all—for a period of some years. Accordingly, a second and larger quantity of irradiated ergosterol has been prepared at the National Institute for Medical Research, and this has been assayed in terms of the original standard preparation. The comparative examination of the new and the original standard preparations has been carried out by eight different laboratories in five different countries, and these eight groups of workers are unanimous in agreeing that the new

standard now available is exactly equivalent to the original standard. There is accordingly now available an adequate amount of this standard preparation of vitamin D to meet the requirements of all workers throughout the world for some years to come. The unit recommended for international use is defined as the vitamin D activity of one milligramme of the international standard solution of irradiated ergosterol.

Vitamin B₁.

The standard for vitamin B₁ recommended for international adoption is a concentrated preparation of the antineuritic vitamin B₁ adsorbed on kaolin. In accordance with the terms of the Conference, this standard has been prepared in the Medical Laboratory, Batavia, Java. The international unit was defined as the antineuritic activity of ten milligrammes of the international standard preparation. The standard preparation is very stable, and, provided it is protected from moisture, appears to retain its activity unchanged.

Suitable supplies of each of the above standards have been sent to approved national institutions for local distribution. In Australia the institution which is in charge of distribution and to which those who wish to obtain supplies of one or other of the standards should apply, is the Commonwealth Serum Laboratories, Parkville, Melbourne, N.2, Victoria.

Obituary.

THOMAS GLEN OLIPHANT.

DR. THOMAS GLEN OLIPHANT, whose death was recorded recently in these pages, was born on January 22, 1889, at Corporate High School, Bendigo, Victoria. He died on May 13, 1933, at Kew, at the residence of his father, to whom he was paying a visit. He first went to school at Corporate High School (his father was headmaster) and later at Saint Andrew's College and the School of Mines, Bendigo. He was captain of his school and played cricket and football. He matriculated in 1908 and entered as a medical student in 1909 at Ormond College, University of Melbourne.

Oliphant graduated in 1914 with honours in medicine, surgery and obstetrics. He became a resident medical officer at the Melbourne Hospital, but had a serious breakdown in health. He became medical officer to the Thirty-Eighth Battalion at Bendigo, but was rejected for service with the army overseas. After acting as *locum tenens* in several places, he bought the practice of Dr. Scott, at Hamilton, Victoria; he remained there till his death. He was a keen and earnest practitioner and achieved a wide popularity by his modesty, his kindness and his devotion to duty. He belonged to several literary societies, and was fond of music. His chief hobbies were cricket and gardening. He is survived by a widow, two sons and a daughter.

Dr. J. Le M. Kneebone writes:

The tragically sudden death of Dr. Oliphant has left a gap which will be very difficult to fill.

Though quiet and unassuming, he was universally popular and found time to take a prominent part in many of the sporting and social activities of Hamilton and district. He had been president of the Hamilton Golf Club and the Hamilton Horticultural Society, and at the time of his death was president of the Bowling Club.

No clearer insight into the character of a man can be obtained than from the intimate association of partnership. He was a most loyal colleague, and honour and sincerity marked all his actions. There was nothing mean or trivial in his nature, and in work as in sport to him the game was always more than the prize.

Ill health had not allowed him to travel, but his outlook and interests were very wide.

His premature death has robbed the community of a man it could ill afford to lose, but his name will not readily be forgotten by the many whom he helped in their time of trouble, as much by his kindness and sympathy as by his skill.

WENTWORTH ROLAND CAVANAGH MAINWARING.

We regret to announce the death of Dr. Wentworth Roland Cavanagh Mainwaring, which occurred on June 27, 1933, at North Adelaide, South Australia.

ALFRED WILLIAM HILL.

We regret to announce the death of Dr. Alfred William Hill, which occurred on June 29, 1933, at Fullarton, Adelaide, South Australia.

Congress Notes.

THE AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

The following appointments as Local Secretaries have been made in the several States of the Commonwealth and in New Zealand.

New South Wales: Dr. J. G. Hunter, British Medical Association House, 135, Macquarie Street, Sydney.

New Zealand, South Island: Dr. G. M. F. Barnett, 83, Stafford Street, Dunedin.

Queensland: Dr. Kenneth Wilson, B.M.A. Building, 35, Adelaide Street, Brisbane.

South Australia: Dr. Alan H. Lendon, North Terrace, Adelaide.

Tasmania: Dr. J. H. B. Walch, 171, Macquarie Street, Hobart.

Victoria: Dr. J. P. Major, Medical Society Hall, Albert Street, East Melbourne.

Western Australia: Dr. L. E. Le Souef, 6, Bank of New South Wales Chambers, St. George's Terrace, Perth.

Intending members of Congress are invited to apply to the Local State or Dominion Secretary for information. Application for membership should be made to the Local Secretary and the application should be accompanied by a remittance of three guineas. Members who do not propose to attend the Congress will pay two guineas; they will receive a copy of the transactions.

Provisional Programme of Congress.

The following provisional programme of Congress has been adopted:

Monday, January 15.

9 a.m. to 4 p.m.—Registration of members at the Tasmanian University Buildings.

8.30 p.m.—Inaugural meeting of Congress at Town Hall, Hobart. Address by Dr. D. H. E. Lines, President of Congress.

Tuesday, January 16.

9.30 a.m. to 12.30 p.m.—All sections. Discussion on cancer and cancer research (Dr. D. H. E. Lines, President, in the chair) at the main hall, Philip Smith Teachers' College, University.

12.30 p.m.—Official photographs to be taken at the University and Philip Smith Teachers' College.

2 p.m. to 4 p.m.—Sectional meetings.

Wednesday, January 17.

9.30 a.m.—Sectional meetings.

2.30 p.m.—Sectional meetings.

3.30 p.m.—Entertainment.

Thursday, January 18.

9.30 a.m.—All sections. "Hospital Problems in Australia".

Dr. D. H. E. Lines in the chair.

2.30 p.m.—Sectional meetings.

8.30 p.m.—Popular lectures.

Friday, January 19.

9.30 a.m. to 12.30 p.m.—Sectional meetings.

2.30 p.m.—Sectional meetings.

Section of Anæsthesia.

The Executive Committee has determined to add to the sections a Section of Anæsthesia. Dr. Cedric Duncombe, Macquarie Street, Hobart, has been appointed Secretary of the Section.

Presidents of Sections.

The following have accepted invitations to act as presidents of sections:

Section of Medicine: Dr. C. Bickerton Blackburn (Sydney).

Section of Surgery: Dr. Bronte Smeaton (Adelaide).

Section of Pathology, Bacteriology and Cancer Research: Dr. J. V. Duhig (Brisbane).

Section of Ophthalmology: Dr. L. Mitchell (Victoria).

Section of Radiology: Dr. H. R. Sear (Sydney).

Travelling Facilities.

Railways.

Members travelling by any State or Commonwealth railways will be entitled to return tickets at single fares plus one-third. This concession also applies to their wives.

In Queensland and Western Australia, sons under the age of sixteen, and unmarried daughters, are also entitled to such reduced fares when accompanying the member.

These concession tickets will be available to enable members to arrive in Hobart seven days before the inaugural meeting, and commence their return journey one month from the opening of Congress, that is, not later than February 15, 1934.

State Secretaries of Congress will be able to give members any information desired.

Shipping.

Members travelling by the Associated Steamship Owners' ships, that is, ships of the Union Steamship Company, Huddart Parker, Limited, Australasian United Steam Navigation Company, Limited, Melbourne Steamship Company, Limited, Adelaide Steamship Company, Limited, and McIlwraith, McEachern, Limited, will be allowed a concession of 10% off their fares. This also applies to their wives. The T.S.S. *Nairana* leaves Melbourne for Launceston on Monday, Wednesday, and Friday, January 8, 10, and 12, 1934, and leaves Launceston for Melbourne on January 23, 25, 27 and 30. The T.S.S. *Loongana* leaves Melbourne for Burnie on Tuesday and Friday, January 9 and 12, 1934.

Members who may desire to break their journey at Launceston or Burnie should communicate with Dr. Grove, the Launceston Public Hospital, for information as to accommodation *et cetera*.

Conditionally on being allowed to carry interstate passengers, and if sufficient inducement offers, the Peninsular and Oriental Steam Navigation Company's R.M.S. *Cathay* would leave Brisbane on January 3, Sydney on January 11, and arrive at Hobart on Saturday, January 13, 1934. The *Mongolia* will leave Fremantle on January 2, Adelaide on January 6, Melbourne on January 8, 1934, arriving at Hobart on Wednesday, January 10. Fourteen days later a ship would call at Hobart to pick up passengers for Melbourne, and one from Melbourne to pick up passengers for Sydney. No concessions are available for members on these overseas ships.

Arrangements have been made by the Executive for a Congress Ship, the *Zealandia*, which will leave Sydney on Friday, January 12, 1934. Accommodation will be available on this ship for 200 persons attending the Congress during the stay of seven days at Hobart. The wharf at which she will berth is within a few minutes' walk of Congress headquarters. The return voyage from Hobart will commence on Sunday, January 21, 1934. The tour, which includes accommodation during the stay at Hobart, will cost £20 per passenger. Those desiring to prolong their stay may book their return by subsequent sailings of the *Zealandia* without additional expense. Intending passengers by this Congress ship should communicate immediately with Dr. E. A. Rogers, 153, Macquarie Street, Hobart.

Trade Exhibition.

Arrangements have been made for holding a trade exhibition in connexion with the Congress. This will provide firms with an opportunity of displaying their wares to the medical profession of Australia and New Zealand. Members of Congress always seize this opportunity of inspecting surgical instruments, dressings, new drugs, books and other equipment. Space will be let at a reasonable rate per square foot. Intending exhibitors are invited to apply as early as possible to Dr. J. S. Reid, 159, Macquarie Street, Hobart.

Congresses.

AMERICAN CONGRESS OF PHYSICAL THERAPY.

THE twelfth annual American Congress of Physical Therapy will be held at Chicago, United States of America, from September 11 to 15, 1933. The programme will include clinical demonstrations and addresses; reports of research will be made. Further information may be obtained from the Executive Secretary, American Congress of Physical Therapy, 30, North Michigan Avenue, Chicago.

Post-Graduate Work.

ANNUAL REFRESHER COURSE IN MELBOURNE.

THE Melbourne Permanent Post-Graduate Committee announces that the annual refresher course will be held in Melbourne from November 13 to 25, 1933. Concurrent with the course, Professor F. Wood Jones, F.R.S., will deliver a special course of six lectures. Further details will be announced at a later date.

At the same time the Melbourne Permanent Post-Graduate Committee proposes to hold a post-graduate course in obstetrics. This course will comprise the general routine work of the Women's Hospital together with special lectures and demonstrations by members of the honorary staff. It is hoped that arrangements may be made for the accommodation in the hospital of those entering the course.

Correspondence.

HAY FEVER AND VASO-MOTOR RHINORRHOEA.

SIR: During recent years many methods of treatment of hay fever and vaso-motor rhinorrhoea have been adopted. Inoculations for suspected allergic conditions have been on trial with but partial success, since for some people at all events, no susceptibility to any particular agent can be discovered. But more recently two methods have obtained some success.

1. The application of carbon dioxide to the nose by inhalation based on the experience of certain European spas does seem to give, at all events, paliative results. Of the accuracy of this statement I have satisfied myself.

2. More recently, zinc ionization of the middle turbinated and the opposed septum has been fairly successful. Dr. Parnell and I have had, in some cases of hay fever, almost dramatic results. Of its utility in rhinorrhoea it is not possible to speak so confidently but amelioration certainly is effected.

Looking back on the history of these most troublesome and unpleasant conditions, it is noteworthy that Francis had some success with cauterization of special parts of the nasal mucous membrane. Whatever be the biological fundamental changes causing hay fever and vaso-motor rhinorrhoea, it seems probable that for practical purposes they must be regarded as local diseases of the nose.

It is noteworthy that time after time good results have been claimed for local treatment. Discounting mistakes of judgement in this respect the repeated return of local treatment is in itself significant of the real nature of those diseases.

Yours, etc.,

JAMES W. BARRETT.

Melbourne,

May 11, 1933.

THE CLOSED DOOR.

SIR: I have to thank Mr. Alan Newton for his reply to me in your journal of June 10. Neither Mr. Newton's letter nor his address to the annual meeting of the College disposes of my implication that the majority of the teachers of this and the rising generations of surgeons in New South Wales would today be ineligible for election to the fellowship of the College. Yet no one would suggest that the Sydney school of surgery has not a history of which it may be proud.

One has nothing but respect and admiration for the aims and ideals of the College in attempting to raise the standard of surgical efficiency, but I ask, with all humility, if it is going the right way to attain its excellent object. Fulfilment of the fellowship regulations may admittedly produce good surgical specialists, although Sir Herbert Maitland thought that the specialist who had not had experience of general practice was often nothing but a trained fool. Is it, then, the standard of specialist practice of surgery which stands most in need of improvement? Rather, I should think that it is the much despised "occasional surgeon" who would benefit most from an intensive educational campaign. If the College wishes to do the greatest service to surgery, let it take these pariahs under its aegis.

Time, distance, and the nature of many surgical diseases are only three factors that make it certain that surgery can never be practised exclusively by specialists. It behoves the College to recognize this and to do something in an official way to encourage and help the man who is far from the metropolitan centres, where rehashes of Samson Wright are served and M.S. degrees are obtained. Perhaps the publication of authoritative articles in journals having a general circulation would be an inexpensive earnest of the College's sincere desire to advance the cause of surgery.

It seems possible that some of the "occasional surgeons" may climb the ladder of experience in the same way that the majority of Sydney's most outstanding surgeons did without ever obtaining a higher qualification. Yet they will be denied the "hall-mark" of the silver vessels and must always remain mere pewter pots. Mr. Newton has not convinced me that there is a royal road to the learning of an art, as the regulations of the College suggest. Before I can admit that my arguments are fallacious, I must be satisfied that the system that produced a Maitland and a Corlette, to mention but two, is quite unworthy in comparison to the "higher degree apprenticeship" system.

Although my ambitions are not surgical, let me assure Mr. Newton that what surgery I have to do is done with all the care and efficiency I command, and in a spirit which, I hope, is in accord with the highest ideals of my profession.

Let me explain, too, that when I signed myself "High Priest" I was being slightly satirical of the attitude which has become so fashionable since a certain noble lord published a treatise on abdominal surgery in America, a country in which the cult of "uplift" is very popular. A spirit of service and *noblesse oblige* is essentially British, but to be constantly talking about it is somewhat foreign.

Yours, etc.,

Queensland,
June 12, 1933.

"HIGH PRIEST."

INTERMITTENT ALCOHOLISM OR DIPSOMANIA.

SIR: May I suggest the importance of finding out if the patient has a *Bacillus coli communis* infection. An examination of the abdomen may reveal enteroptosis, an old diseased appendix, intestinal adhesions, kinks, chronic constipation et cetera. These infections may occur at intervals and clear up quickly, but recur for many years, the alcoholic bouts being connected with a bacillæmia. I have seen a number of cases of this nature.

Yours, etc.,

J. MORRIS ROE.

Victory Chambers,
Queen Street,
Brisbane,
June 29, 1933.

UNUSUAL HÆMOPTYSIS: AN INQUIRY.

SIR: I wish to report the following case, as it contains one feature of exceptional interest.

Two years ago I first saw the patient, a female, aged twenty-six, who was suffering from an ischio-rectal abscess. This was drained. A radiograph and bacteriological examination of the sputum followed, both of which disclosed the presence of pulmonary tuberculosis. Treatment proved to be difficult on account of the development of an anal fistula, which eventually healed completely after three months of surgical treatment. The customary outdoor life was carried out by the patient, and she has made a very satisfactory recovery. Her weight is now twelve stone, she eats well, can do light house work comfortably, and has but a slight cough.

The point of exceptional interest to which I referred above is that during each menstrual period she coughs up a small quantity of blood. This occurs within a few hours of the beginning of her periods and lasts until the day the bleeding *per vaginam* ceases. She feels slightly off colour, but not more than one would expect during such a time. On these occasions her lung condition does not seem to get any worse. The patient has a retroverted uterus.

At first I thought that the fact of the patient being unwell may have lowered her resistance and thus caused the hæmoptysis, but seeing that this is not accompanied by any symptoms or signs of increased lung disease it appears more likely that this may be a case of vicarious menstruation.

I would be very pleased to hear if any of your readers have had similar cases, and also if this bleeding from the lung has any significance as far as the progress of the tubercular condition is concerned.

Yours, etc.,

S. GOLDBERG.

Griffith,
New South Wales,
June 30, 1933.

ACHLORHYDRIC ANÆMIA.

SIR: There has been very great interest of late in the subject of anæmia. The wonderful effects of liver feeding in pernicious anæmia patients are now commonplace, and the great value even in severe cases of intramuscular injections of liver extract is established.

In hypochromic or "simple" achlorhydric anæmia the response to treatment with iron preparations is no less remarkable. Treatment is easy, but the diagnosis of this type of anæmia is not always clear. Secondary anæmia from any cause, and especially the anæmia associated with cancer of the stomach, may resemble it very closely. A few weeks' treatment with iron may appear encouraging; yet, far from establishing the diagnosis of achlorhydric anæmia, this very circumstance may actually mislead the physician.

Two of my recent cases illustrate the point.

The first a woman of forty-eight years, was sent to me by a colleague, with the diagnosis of gastric cancer. This diagnosis proved to be correct, but although I concurred with it at first, I was led to doubt it for a time by the striking response to treatment. The woman was very wasted, even cachectic, and too ill to walk or sit up. No mass could be felt in the abdomen. The X ray findings were inconclusive. Fractional meal showed absence of hydrochloric acid. Blood examination showed anæmia of the hypochromic type, with the hæmoglobin percentage of 55. The finger-nails were the best example of the spoon form (koilonychia) I have yet seen. A surgical colleague agreed with the view that achlorhydric anæmia might explain the whole condition, and advised against exploratory operation. After eight weeks' treatment with iron and hydrochloric acid the hæmoglobin percentage had reached 75, the weight had increased by over a stone, the patient was enjoying ordinary full diet and was well enough to sit out of bed and even walk a little. But progress was not maintained; she began to fail rapidly and seven weeks later she died. Irregular and often high pyrexia, increasing general weakness and abdominal discomfort, enlargement of the liver, and intense obstructive jaundice were the terminal events. *Post mortem* investigation showed general malignant infiltration of the liver. No primary growth was found. The examination was done under restricted conditions and the search was incomplete; a small cancer of the stomach was probably overlooked.

My second patient, a woman of fifty-three years, had weakness and vague digestive discomfort of the flatulent type for a year. Again the symptoms and the blood picture led me to diagnose achlorhydric anæmia, again the response to treatment was very encouraging, and again I have had to alter my view. In two months the hæmoglobin percentage rose from 50 to 73, and the general health improved. But the digestive discomfort remained. Opaque meal examination was then done; it showed a large five-hour residue in the stomach and irregular outline of the pyloric end. A definite mass is now to be felt. Pallor and asthenia are returning. The cause of her trouble is no longer in doubt. It is well to bear in mind this mimicry of "simple" achlorhydric anæmia by cancer of the stomach, and not in every case to be buoyed up by the results of treatment. Initial improvement may occur, but not be maintained. Whereas in pernicious anæmia the result of adequate liver feeding clinches the diagnosis, the effects of iron administration in a supposed case of "simple" achlorhydric anæmia may be inconclusive and misleading. The need for full examination and for care in evaluating the progress is obvious.

Yours, etc.,

A. R. SOUTHWOOD.

Adelaide,
July 3, 1933.

THE TONSIL PROBLEM.

SIR: In today's journal Dr. Kent Hughes says: "Today I am convinced of the certainty of curing enlarged tonsils and adenoids by removing the sinusitis".

My experience in some thousands of children has been such that today I am convinced of the probability of curing sinusitis if I remove enlarged tonsils and adenoids.

Now, Dr. Hughes's certainty excels my probability and I am prepared to mend my ways in pursuit of it. Would

he be good enough to say what sinuses he has found affected and what measure he applies to them severally to secure his excellent results in children up to the age of fourteen years?

Yours, etc.,
ARTHUR MURPHY.

Brisbane,
July 15, 1933.

THE LISTERIAN ORATION.

SIR: In Dr. F. A. Maguire's Listerian Oration, appearing in your journal of July 15, there appears the following sentence: "Gonorrhoea is a preventible disease, and as the late King Edward said, 'If preventible, why not prevented'". This would surely convey the impression that the late King Edward was referring in his remark to this venereal disease. Actually, of course, His late Britannic Majesty was speaking about tuberculosis when he uttered this now historic aphorism.

Yours, etc.,
COTTER HARVEY.

British Medical Association House,
137, Macquarie Street,
Sydney,
July 17, 1933.

AN ECONOMIC COMMENTARY AND DIAGNOSIS.

SIR: "M.D., C.M.'s" letter on the so-called Douglas Credit System makes very clear the obvious and elementary fallacies in the latter gentleman's revival of an ancient example of loose thinking.

There is no doubt that the world is economically sick, but there is no more justification for introducing a quack remedy unsupported by any basis of pathology in the case of an economic than a physical illness. As a medical man, "M.D., C.M." should be as loath to apply dangerous and absurd treatment to economic disturbances as to one of his own patients. It is incredible that any person who has had a scientific training could accept the childish statements and obvious fallacies of the A plus B theorem.

If this theorem were true, it is obvious that no one employed in primary industry could have at any time in history received payment of any kind, because according to Major Douglas there has never been sufficient purchasing power, money, credit, wealth, A factor, or what you will, to pay him. If this theorem were true now, it has necessarily been always true. If it has always been true, one of several things must have been going on without our knowledge. Either the world has been producing since there have been any secondary industries, goods for which it has been impossible to find purchasers, or the proprietors of secondary industries have successfully evaded the payment of taxes, bank charges *et cetera*, or for the raw material consumed in the industry. If the first is true, there must be in existence somewhere enormous quantities of commodities manufactured since the time of Julius Caesar, which, if we believe Major Douglas, cannot possibly have been purchased. If these tons of merchandise have not been purchased and consumed, where are they now? Any intelligent person prefers to believe that they have been bought and used in which case the A plus B theorem of necessity is false. It is almost as ridiculous to ask us to believe that manufacturers have not paid taxes, bank charges and other obligations than wages.

The truth is that Major Douglas, ignorant of the principles of political economy and of the elements of logic, has, in all sincerity, propounded an utterly ridiculous theory, the fallacy of which has been demonstrated by every committee of experts that has considered it, and becomes obvious to any person who is accustomed to think logically.

If the matter rested here and the idea was only taken up by harmless cranks of the type that adopt health fads and join "anti" societies, we could afford to dismiss the matter with a tolerant smile, but unfortunately the theory

has been seized upon by the worst types of demagogues and by those whose aim it is to destroy our civilization. Many of the phrases used by "M.D., C.M." suggest that he has been reading without proper concentration the very type of book and pamphlet that is being circulated among the unthinking by foreign organizations. Such expressions as "spider-web of Wall Street", "money Moloch's lions" *et cetera*, have no appeal to educated men, but are freely used in the Domain. These phrases accompanied by the time-honoured method of imputing bias to the committees of investigation are typical of a variety of orator, whom we recognize as hostile to civilization and entirely destructive in his aims.

If the Douglas Credit System were even in part true, it would be welcomed by every economist and most governments as an easy way out of our present troubles. Unfortunately for its disciples, it has been recognized at sight as a rather clumsy method of uncontrolled inflation, and we have had too many recent examples of that line of treatment for even the lowest elements in our midst to swallow it without the gilding supplied by the use of "A plus B", "Abracadabra" or other mysterious but meaningless phrases.

In actual fact there is no easy way out of our present difficulties. This is not the time to hand over the patient to charlatans or cranks, and we must find our way back to health by hard work and intelligence and by taking the advice of experts rather than quacks, no matter how good their intentions.

Yours, etc.,
H. C. E. DONOVAN.

195, Macquarie Street,
Sydney.
Undated.

Proceedings of the Australian Medical Boards.

TASMANIA.

The undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Bryan, Francis John, M.B., B.S., 1928 (Univ. Melbourne), Public Hospital, Launceston.
Cotter, Timothy John, M.B., B.S., 1924 (Univ. Melbourne), Public Hospital, Launceston.

Corrigendum.

THE TREATMENT OF DIABETES MELLITUS.

AN error has occurred in the special article on the treatment of *diabetes mellitus*, by Dr. Kempson Maddox, which appeared in the issue of July 22, 1933. On page 122, in the second column, under the heading "Coma", the sentence: "Insulin should be administered by mouth or intravenously" should obviously read: "Glucose should be administered by mouth or intravenously."

Books Received.

NON-TROPICAL SPRUE: A STUDY IN IDIOPATHIC STEATORRHOEA, by T. E. H. Thaysen, M.D.: 1932. Copenhagen: Levin and Munksgaard; London: Humphrey Milford. Crown 4to., pp. 258, with illustrations. Price: 12s. 6d. net.

VARICOSE VEINS AND HEMORRHOIDS AND THEIR TREATMENT, by V. Meisen, M.D., with preface by A. Krogh, Ph.D.: 1932. Copenhagen: Levin and Munksgaard; London: Humphrey Milford. Demy 4to., pp. 149, with illustrations. Price: 10s. 6d. net.

THE TREATMENT OF FUNCTIONAL NERVE CASES BY THE METHOD OF NEURO-INDUCTION: AN ESSAY, by L. Inkster, M.A., M.R.C.S.; 1933. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 80. Price: 3s. 6d. net.

A NEW APPROACH TO DIETETIC THERAPY, by E. Földes, M.D.; 1933. Boston: Richard G. Badger. Royal 8vo., pp. 446. Price \$5.00 net.

MINOR MONOGRAPH SERIES: COLDS AND HAY FEVER, by F. Coke, F.R.C.S.; 1933. London: Baillière, Tindall and Cox. Crown 8vo., pp. 158. Price: 5s. net.

A STUDENT'S MANUAL OF BIRTH CONTROL, by L. C. Butler, M.R.C.S., L.R.C.P., D.P.H.; 1933. London: Noel Douglas. Crown 8vo., pp. 39. Price: 1s. 6d. net.

THE ADJUSTMENT OF MUSCULAR HABITS, by J. K. McConnel, with foreword by W. E. Le Gros Clark; 1933. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 141, with illustrations. Price: 4s. 6d. net.

RECENT ADVANCES IN RADIUM, by W. R. Ward, M.B., B.S., M.R.C.S., and A. J. D. Smith, M.B., B.S., M.R.C.S.; 1933. London: J. and A. Churchill. Royal 8vo., pp. 324, with illustrations. Price: 21s. net.

URINE AND URINALYSIS, by L. Gerahenfeld, Ph.M., B.Sc., P.D.; 1933. Philadelphia: Lea and Febiger. Crown 8vo., pp. 288, with 38 illustrations.

THE MIND IN DAILY LIFE, by R. D. Gillespie, M.D., M.R.C.P., D.P.M.; 1933. London: Methuen and Company, Limited. Crown 8vo., pp. 296, with 9 diagrams. Price: 5s. 6d. net.

IDOLS AND INVALIDS, by J. Kemble, Ch.M., F.R.C.S.; 1933. London: Methuen and Company, Limited. Crown 8vo., pp. 221. Price: 6s. net.

DISEASES OF THE HEART DESCRIBED FOR PRACTITIONERS AND STUDENTS, by Sir Thomas Lewis, C.B.E., F.R.S., M.D., D.Sc., LL.D., F.R.C.P.; 1933. London: Macmillan and Company, Limited; Australia: Angus and Robertson. Royal 8vo., pp. 317. Price: 19s. net.

CLIO MEDICA: A SERIES OF PRIMERS ON THE HISTORY OF MEDICINE, edited by E. B. Krumbhaar, M.D. IX: Medicine in Canada. Folscap 8vo., pp. 150, with illustrations. Price: \$1.50 net.

SEX IN MODERN LIFE: A SURVEY OF SEXUAL LIFE IN ADOLESCENCE AND MARRIAGE, by R. V. Storer; 1933. Australia: James Little and Son. Crown 8vo., pp. 227, with illustrations. Price: 12s. 6d. net.

NATURAL CHILD BIRTH, by G. D. Read, M.A., M.D.; 1933. London: William Heinemann (Medical Books) Limited. Demy 8vo., pp. 136. Price: 7s. 6d. net.

THE CURE OF HÆMORRHOIDS, VARICOSE VEINS AND ULCERATION AND ALLIED CONDITIONS BY MODERN METHODS OF INJECTION AND BANDAGING, by S. McAusland, B.A., M.D., Ch.B., M.R.C.S., L.R.C.P.; 1933. London: John Bale, Sons and Danielsson, Limited. Demy 8vo., pp. 69, with illustrations. Price: 3s. 6d. net.

WOMAN'S PERIODICITY, by M. Chadwick, S.R.N.; 1933. London: Noel Douglas. Crown 8vo., pp. 225. Price: 6s. net.

STUDIES ON THE PHYSIOLOGY OF THE EYE: STILL REACTION, SLEEP, DREAMS, HIBERNATION, REPRESSION, HYPNOSIS, NARCOSIS, COMA, AND ALLIED CONDITIONS, by J. G. Byrne; 1933. London: H. K. Lewis and Company, Limited. Royal 8vo., pp. 428, with 48 illustrations. Price: 40s. net.

Medical Appointments.

Dr. R. M. Dunstone (B.M.A.) has been appointed Government Medical Officer at Bogan Gate, New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought etc., see "Advertiser", pages xvi, xvii

AUSTRALASIAN MASSAGE ASSOCIATION, SYDNEY, NEW SOUTH WALES: Lecturers.

DIRECTOR-GENERAL OF PUBLIC HEALTH, SYDNEY, NEW SOUTH WALES: Honorary Officers.

HOBART PUBLIC HOSPITAL, HOBART, TASMANIA: Junior Resident Medical Officers.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.

MELBOURNE HOSPITAL, MELBOURNE, VICTORIA: Honorary Officers.

THE OTAGO HOSPITAL BOARD, DUNEDIN, NEW ZEALAND: Resident Surgical Officer.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phoenix Mutual Provident Society.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Western Australian: Honorary Secretary, 65, Saint George's Terrace, Perth.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-3.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 6s. abroad per annum payable in advance.